Dissertation on

"A COMPARITIVE STUDY TO ANALYSE THE SIGNIFICANCE OF PRESSURE-TO-CORNEA INDEX IN PRIMARY OPEN ANGLE GLAUCOMA , NORMAL TENSION GLAUCOMA , OCULAR HYPERTENSIVE PATIENTS AND PATIENTS WITHOUT GLAUCOMA"

Submitted in partial fulfillment of requirements of

MASTER OF SURGERY DEGREE

BRANCH – III – (OPHTHALMOLOGY)

GOVT. RAJAJI HOSPITAL, MADURAI MEDICAL COLLEGE

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THE TAMILNADU

Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI

CERTIFICATE

This is to certify that this dissertation entitled **"A COMPARITIVE STUDY TO ANALYSE THE SIGNIFICANCE OF PRESSURE-TO-CORNEA INDEX IN PRIMARY OPEN ANGLE GLAUCOMA , NORMAL TENSION GLAUCOMA , OCULAR HYPERTENSIVE PATIENTS AND PATIENTS WITHOUT GLAUCOMA"** is a bonafide record of research work done by **Dr.P.ARUNKUMAR,** Post Graduate Resident in Department of Ophthalmology,Madurai Medical College, Madurai.

He has submitted this in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University, for the award of Master of Surgery Degree Branch III (Ophthalmology), under our guidance and supervision during the academic years 2016-2018.

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This is to certify that this dissertation entitled **"A COMPARITIVE STUDY TO ANALYSE THE SIGNIFICANCE OF PRESSURE-TO-CORNEA INDEX IN PRIMARY OPEN ANGLE GLAUCOMA , NORMAL TENSION GLAUCOMA, OCULAR HYPERTENSIVE PATIENTS AND PATIENTS WITHOUT GLAUCOMA** is a bonafide record of research work done by **Dr. P.ARUNKUMAR,** Post Graduate Resident in Department of Ophthalmology, Madurai Medical College, Madurai.

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DECLARATION

I, **Dr. P. ARUNKUMAR**, hereby solemnly declare that this dissertation titled **"A COMPARITIVE STUDY TO ANALYSE THE SIGNIFICANCE OF PRESSURE-TO-CORNEA INDEX IN PRIMARY OPEN ANGLE GLAUCOMA , NORMAL TENSION GLAUCOMA, OCULAR HYPERTENSIVE PATIENTS AND PATIENTS WITHOUT GLAUCOMA"** was done by me.

I also declare that this bonafide work / a part of this work was not submitted by me / anyone else, for any award, for Degree / Diploma to any other University / Board either in India / abroad. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of Master of Surgery degree Branch -III (Ophthalmology) to be held in May 2018.

Place: Madurai **(DR.P.ARUNKUMAR)**

Date:

.

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INDEX

PART ONE

INTRODUCTION

Normal human central corneal thickness varies between a range of *490 μm to 560 μm.* Whereas the intraocular pressure measured by the gold standard method **'Goldmann Applanation Tonometry'** is based on the assumption that CCT is 520 μm.

The measured intraocular pressure becomes falsely high or falsely low when measured on thicker corneas or thinner corneas respectively. So, IOP has to be adjusted according to the central corneal thickness by a correction factor.

Whereas the relationship between IOP and CCT is not linear. So even if the correction factor is applied, the correction of IOP over the extreme values of CCT becomes inaccurate and not reliable. Also, none of the correction factors, so far proposed, has been universally accepted as a standard formula.

So, to overcome this error in correction of IOP by various nonstandardized formulae, and also to integrate IOP and CCT as a single risk factor for glaucoma, a new index called as **Pressure-To-Cornea Index(PCI)** was introduced.

"PCI is the ratio between the highest recordable pretreatment IOP in mm Hg to the cubic power of Central Corneal Thickness(CCT) expressed in mm."

PCI = Pretreatment IOP (mm Hg) CCT^3 (mm)

e.g., if CCT is 545 μm and measured untreated IOP is 18 mmHg, then PCI = 18/ (0.545 x 0.545 x 0.545)

= 18 / 0.161878625 = **111.2**

GLAUCOMA

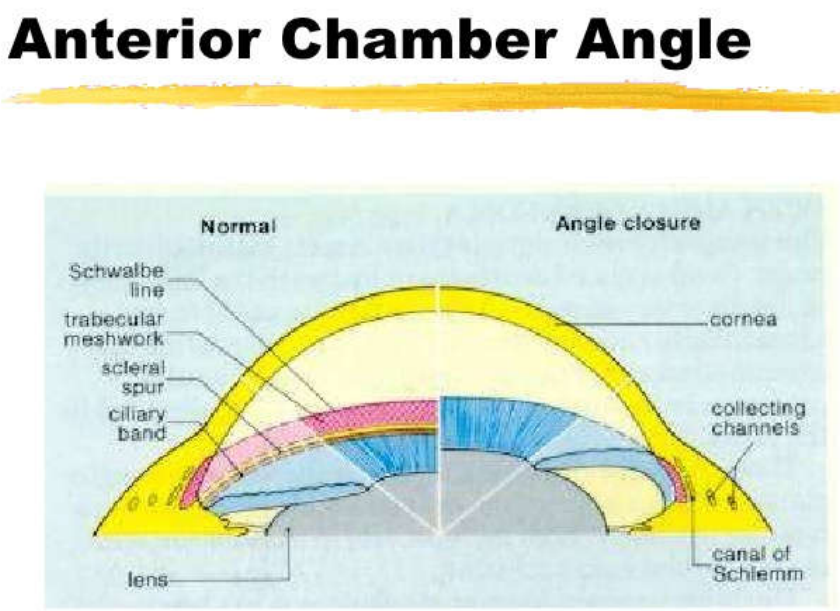
Glaucoma is defined as a multifactorial chronic progressive optic neuropathy caused by a group of ocular conditions ,causing damage to retinal ganglion cells at levels beyond normal baseline ager related loss resulting in optic disc changes and corresponding visual field defects with raised intraocular pressure being the only modifiable risk factor.

ANATOMICAL CONSIDERATIONS:

Aqueous humour is secreted by the ciliary body in the posterior chamber by diffusion, ultrafiltration & secretion. Aqueous leaves the posterior chamber via pupil & reaches the anterior chamber, from where it is drained through the angle of anterior chamber.

ANGLE OF THE ANTERIOR CHAMBER:

Plays an important role in aqueous drainage. Clinically it can be visualised by using gonioscopy. From posterior to anterior, angle recess is formed by following structures:



1) THE CILIARY BAND:

Formed by anterior most part of the ciliary body between its attachment to scleral spur & iris insertion. Appears as grey or dark brown band. Width of the ciliary band depends on level of iris insertion, hence tends to be wider in myopes & vice versa in hypermetropes.

2) SCLERAL SPUR:

It is the posterior portion of scleral sulcus seen as a prominent white line on gonio. Posterior to this is the ciliary body & corneoscleral meshwork anteriorly.

3) TRABECULAR MESHWORK:

Seen as a band anterior to scleral spur. Has no pigmentation at birth & pigment develops with increasing age. Has 3 parts namely inner uveal meshwork, middle corneoscleral meshwork & outer juxtacanalicular meshwork. Outer portion offers the normal resistance to aqueous outflow & it connects the corneoscleral meshwork with the schlem's canal.

It has three parts.

1. Uveal meshwork :

It is the innermost part of trabecular meshwork and extends from the iris root and ciliary body to the Schwalbe's line. The trabeculae of uveal meshwork are cord like, two to three layers thick. The arrangement of uveal trabecular bands creates irregular openings which vary in size from 25μ to 75 μ .

On electron microscopy, each trabeculae is seen to have three concentric layers, a central collagenous core, a middle layer of a basement membrane like material with a periodicity of 1000^{o}A; and enclosing trabecular cells.

ii) Corneoscleral meshwork

It forms the larger middle portion and extends from the scleral spur to the laeral wall of the scleral sulcus. It consists of flat sheets of trabeculae which are perforated by elliptical openings which are smaller than those in the Uveal meshwork (5-50μ). These holes become progressively smaller as the trabecular sheets approach Schlemm's canal and holes of deepest layer may be even 1-2μ in size. Electron microscopic structure of each trabeculae is similar to that of trabeculae of uveal meshwork.

iii) Juxtacanalicular (endothelial) meshwork

It forms the outermost portion of the trabecular meshwork. It is that part of the trabecular meshwork. It is that part of the trabecular meshwork which mainly offers the normal resistance to aqueous outflow. It consists of a layer of connective tissue (containing 2-5 layers of loosely arranged cells, embedded in an extracellular matrix) lined on either side by endothelium. This narrow part of trabeculum (thickness 2-20μm) connects the corneoscleral meshwork with Schlemm's canal. In fact the outer endothelial layer of the juxta canalicular meshwork comprises the inner wall of Schlemm's canal and the inner endothelial layer of the juxta-canalicular meshwork becomes continuous with the endothelium of corneoscleral meshwork.

Schlemms canal is the endothelial lined oval channel present circumferentially in the scleral sulcus. Outer wall has numerous openings of the collector channels. It has numerous septae which passes from the collector channels to inner wall of schlem's canal.

4) SCHWALBE"S LINE: Formed by prominent podein end of Descemets Membrane of cornea. Forms the anterior most part of angle structures, seen as a white band. Corneal wedge is used to identify the schwalbes line.

SHAFFER'S GRADING SYSTEM OF ANGLES

GRADE	ANGLE STRUCTURES VISIBLE
GRADE 4 - 35-45 deg, widest	Upto ciliary body is visible
GRADE 3 - 25-35 deg, open angle	Scleral spur seen
GRADE 2 - 20 deg, moderately narrow	Upto trabecular meshwork can be seen
GRADE 1 - 10deg, very narrow angle	Only schwalbe's line can be seen
SLIT ANGLE	No angle structures are visible without obvious iridotrabecular contact.
GRADE 0 - 0 deg	Closed angle due to iridotrabecular contact.

2. SCHELE'S GONIOSCOPIC CLASSIFICATION

S.No.	Grade	Structure visible
1.	Wide open	All structure visible
2.	Grade 1 narrow	Hard to see the root of iris
3.	Grade 2 narrow	Ciliary body band oscured
4.	Grade 3 narrow	Posterior trabecular meshwork obscured
5.	Grade 4 narrow	Only Schwalbe' line visible

3. SPAETH CLASSIFICATION

Takes Four Parameters Into Consideration

a. Site of iris root insertion

A	Anterior of TM (i.e. Schwalbe's line)
B	Behind schwalbe's line (at the level of TM)
C	Centred at the level of scleral spur
D	Deep to scleral spur (i.e. anterior to CB)
E	Extremely deep inserted into CB

b. Width or geometric angle of iris insertion

The angle between the intersection of imaginary tangents formed by peripheral third of iris and the inner wall of corneosclearal junction. It is graded as 10, 20, 30 degree and 40 degree.

b. Contour of peripheral iris near the angle

S	Steep or convex configuration
R	Regular or flat
Q	Queer – deeply concave

c. **Intensity of trabecular meshwork pigmentation**:
minimal or no pigment grade to dense pigment deposition grade 4.

CLASSIFICATION OF GLAUCOMA

1. CONGENITAL/DEVELOPMENTAL

2. ACQUIRED

a. OPEN-ANGLE GLAUCOMA

i. PRIMARY

1. Primary open angle glaucoma

2. Normal tension glaucoma

ii. SECONDARY

1. Corticosteroid – induced glaucoma 2. Pigmentary glaucoma

3. Exfoliation glaucoma

4. Inflammatory glaucoma

5. Post—traumatic

6. Neovascular glaucoma (early stage)

7. Ghost cell glaucoma

8. Hemosiderotic glaucoma

9. Angle recession glaucoma

10.Lens-protein glaucoma

11.Lens-particle glaucoma

12.Phacoanaphylactic glaucoma

13. Increased episcleral venous pressure 14.Masquerade (tumour)

b. ANGLE-CLOSURE GLAUCOMA

i. PRIMARY

1. With pupillary block - Acute,subacute, chronic 2. Without pupillary block – plateau iris

ii. SECONDARY

1. With pupillary block

a. Inflammatory(with seclusion pupillae or occlusion pupillae)

b. Phacomorphic c. Siliconeoil

d. Vitreous block

2. Without pupillary block

a. Neovascular glaucoma (late stage)

b. Iridocorneal endothelial syndrome

c. Aqueousmisdirectionsyndrome

d. Epithelial downgrowth

e. Fibrousingrowth

c. MIXED

PRIMARY OPEN ANGLE GLAUCOMA: Is a chronic ischemic progressive optic neuropathy in adults characterized by accelerated ganglion cell death and subsequent axonal loss. This is seen as progressive glaucomatous optic disc

changes, glaucomatous optic atrophy and corresponding characteristic visual field loss.

This is characterized by

1. Raised iop > 21mmHg
2. Open anterior chamber angle on gonioscopy
3. Characteristic Visual field defects
4. Evidence of glaucomatous optic nerve head damage on fundus examination

Absence of other secondary causes of OAG.

NORMAL TENSION GLAUCOMA:

It is variant of POAG, characterized by

1. A mean iop <_ 21mmhg on diurnal testing
2. Glaucomatous optic disc damage.
3. Open drainage angle on gonioscopy
4. Absence of secondary causes for glaucomatous optic disc damage.
5. Visual field loss.

OCULAR HYPERTENSION:

Characterized by

1. Raised iop >21mmhg.
2. Absence of glaucomatous optic nerve head damage
3. Absence of visual field defects
4. Open angles on gonioscopy

INTRAOCULAR PRESSURE

“Intraocular pressure (IOP) is defined as the pressure exerted by the intraocular contents on the coats of the eyeball”. It is the most important and only modifiable risk factor for glaucoma. However, glaucoma can occur even with normal IOP.

Normal IOP is the IOP which does not lead to any glaucomatous damage to the optic nerve head and is in the range of 10 to 21 mm Hg. **Normal diurnal variation in IOP** is 3 to 6 mm Hg. IOP > 21 mm Hg or diurnal variation more than 8 mm Hg even with normal IOP becomes a risk factor and raise the suspicion of glaucoma.

METHODS TO MEASURE IOP

Tonometry is the method of measuring IOP and the instrument used is called as tonometer. Tonometer can be

1) Indentation tonometry

(a) Schiotz

(b) Herrington

(c) Grants

(d) Maurice

2) Applanation tonometry

i) <u>Variable force</u>

(a) Goldmann

(b) Perkins

(c) Draeger

(d) Mackay-Marg

ii) Variable area

(a) Maklakov – Kalfa

(b) Applanometer

(c) Tonomat

(d) Halberg

iii) Non contact tonometry –three newer modalities

a) Ocular response analyzer

b) Dynamic contour tonometry(pascal) c) Rebound tonometer

d) Transpalpabrel tonometer

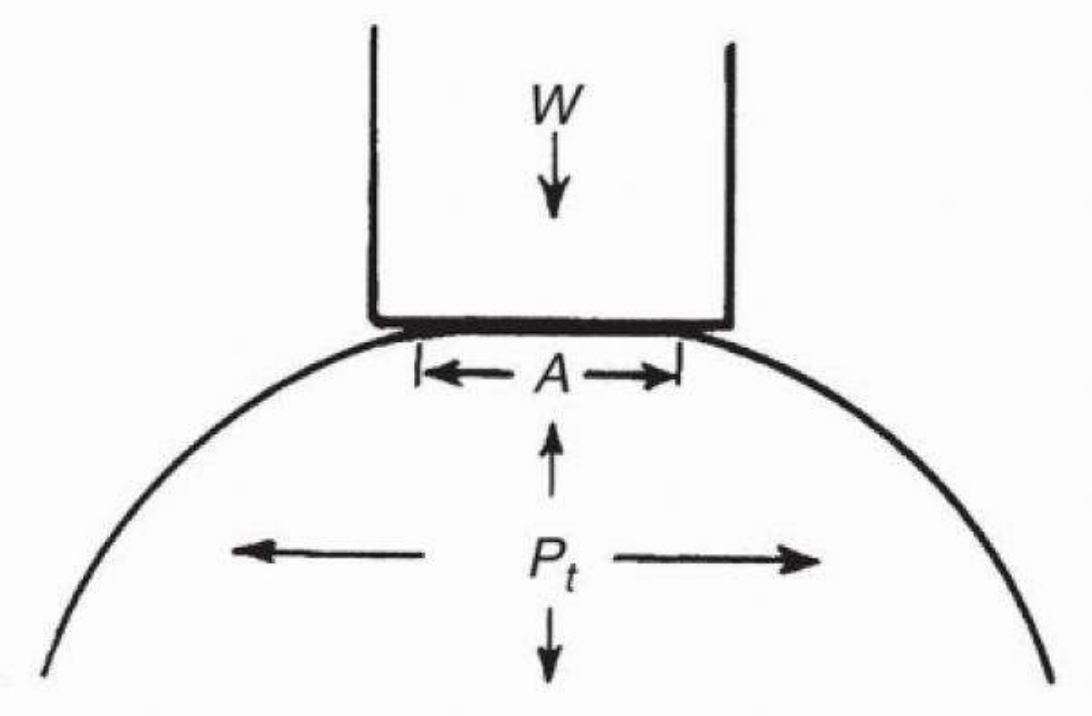
GOLDMANN APPLANATION TONOMETRY (GAT)

- Of these, **Goldmannapplanationtonometer is considered as the GOLD STANDARD** method to measure IOP, since it is reliable and accurate, reproducible and not influenced by scleral rigidity.

- It is a constant-area applanation tonometer and determines the force necessary to flatten (or applanate) a 3.06 mm diameter area of the cornea. Also, there is minimal displacement (0.5 μl) of fluid or minimal increase in IOP with applanation, thus it is unaffected by scleral rigidity.

- It is based on the modified **Imbert-Fick's law**, also called as Maklakoff- Fick's law.

- Original Imbert-Fick's law states that "the force (**W**) against a perfectly flexible, dry, infinitely thin, perfect sphere is equal to the pressure(**Pt)** inside the sphere multiplied by the area of flattening (**A**) by the external force". **$W=P_tX\ A$**



SPHERE ususally obeys to Imbert-Fick law.

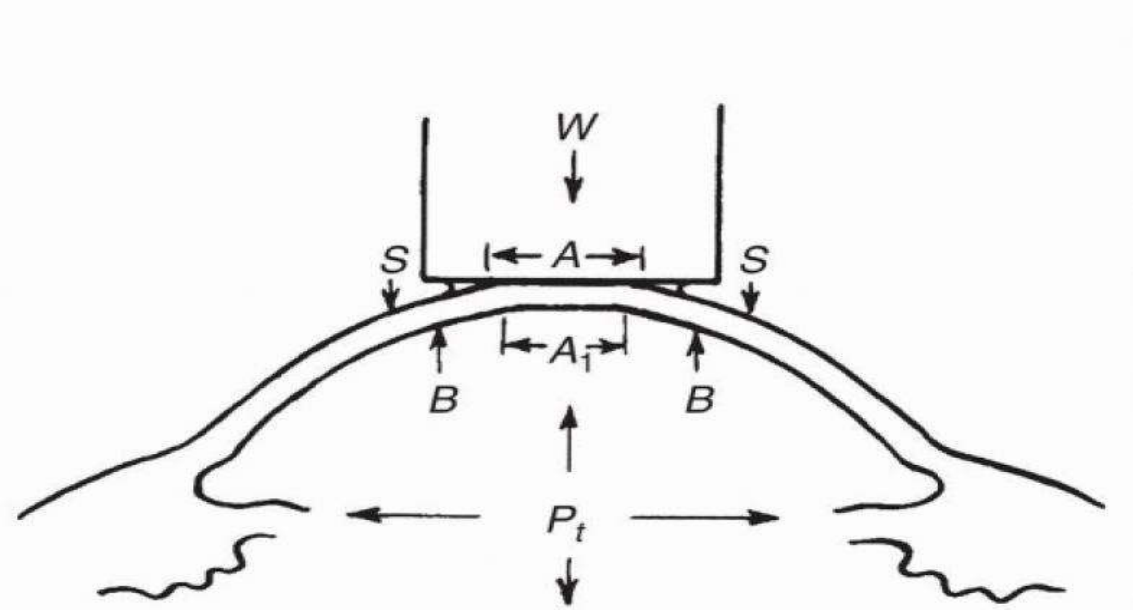
But cornea is aspherical, wet, not perfectly flexible, not infinitely thin. So, cornea fails to obey Imbert-FiCK LAW AND ADDITIONAL FORCES COME TO ACTION.

1. The surface tension (S) of the tear film which acts towards the cornea.

2. the force (B) offered by the cornea to the applanating surface away from the eye due to lack of flexibility, which is independent of the intraocular pressure. Also, since cornea has thickness of about 550 μm, the outer area (A) of corneal flattening differs from the

inner area of flattening (A1). Only flattening of inner corneal area (A_1) is considered as

important. So, the Imbert-Fick's law gets modified as **$W+S = P_t A_1 + B$**

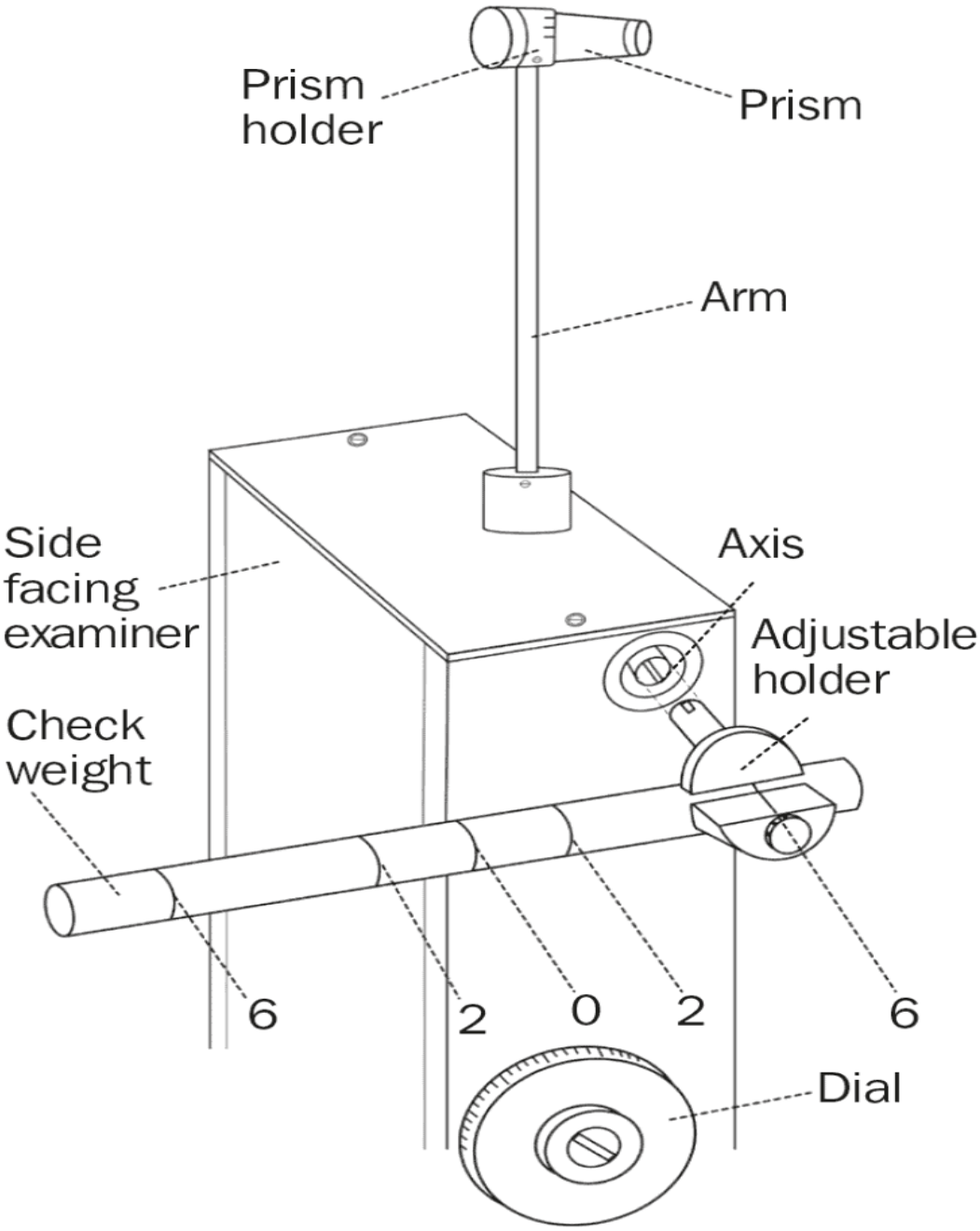


But when **A_1**= 7.35 mm^2, A = 3.06 mm and the central corneal thickness is assumed to be 520 μm, the additional forces S and B are equal and get balanced with each other and W becomes equal to P_t. So the external area of applanation is kept constant as 3.06 mm in the standard instrument.

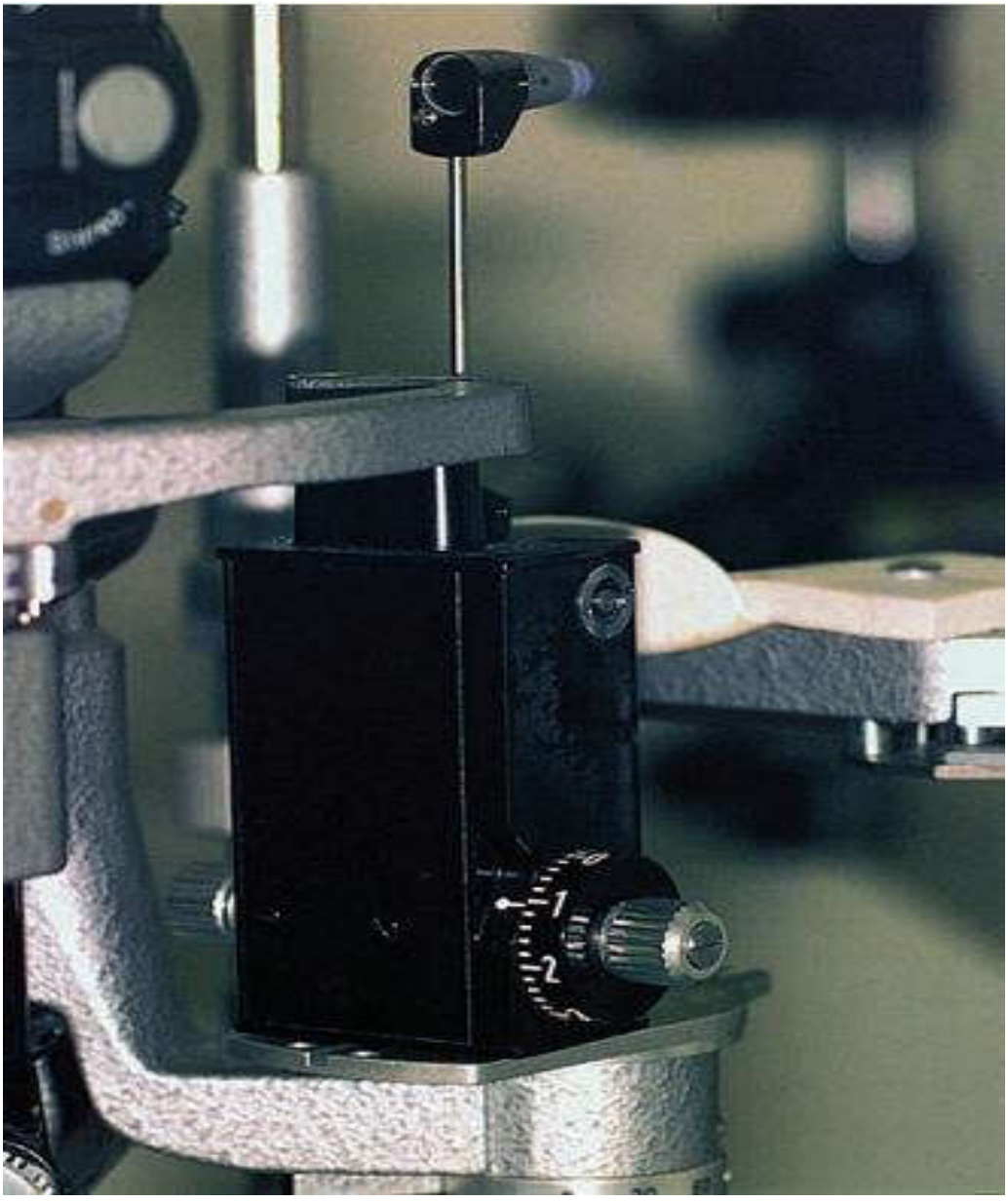
Whereas the normal central corneal thickness varies between a range of 490 μm to 560 μm. So, the measured intraocular pressure is not accurate for the corneal thickness. It is false high on thicker corneas as in ocular hypertension or deposition of any additional tissue; false low on thinner corneas as in normal tension glaucoma, following keratorefractive surgery. So, IOP measure by GAT has to be adjusted according to the central corneal thickness by a correction factor.

GAT - INSTRUMENT :

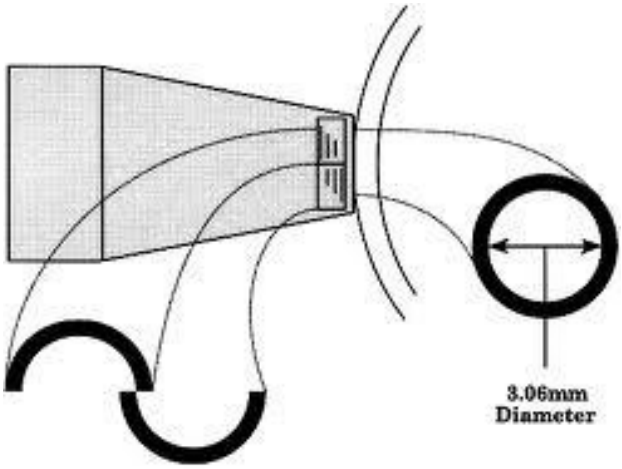
- The instrument is mounted on a standard slit lamp. It contains aapplanating unit which is attached by a rod, to a housing with levers to adjust the force of the biprism against the cornea.



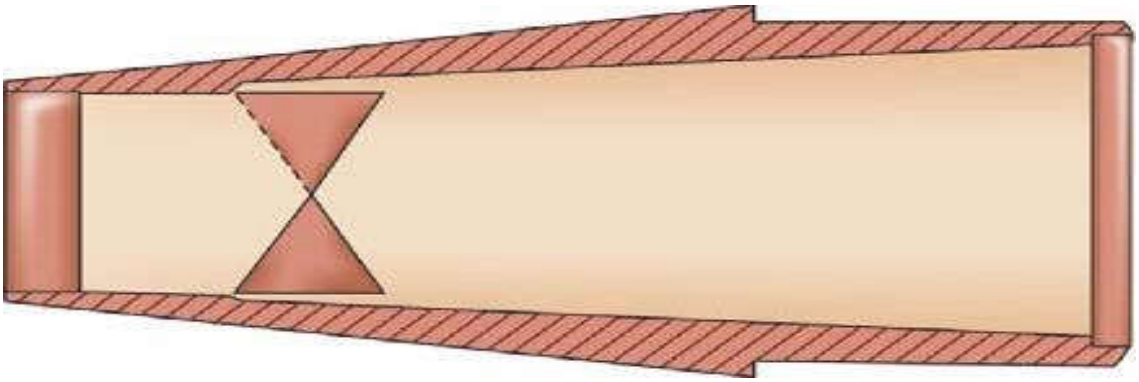
- Applanating unit contains two beam splitting prisms which optically convert circular area of corneal contact into 2 semicircles



- Tension knob with markings, attached to the housing below, is used to adjust the force for applanation.



Optical Endpoint



TECHNIQUE OF MEASUREMENT:

- The applanation unit is set in correct position on the slit lamp.

- One drop of a topical anesthetic - **0.5% proparacaine**, is instilled in each eye

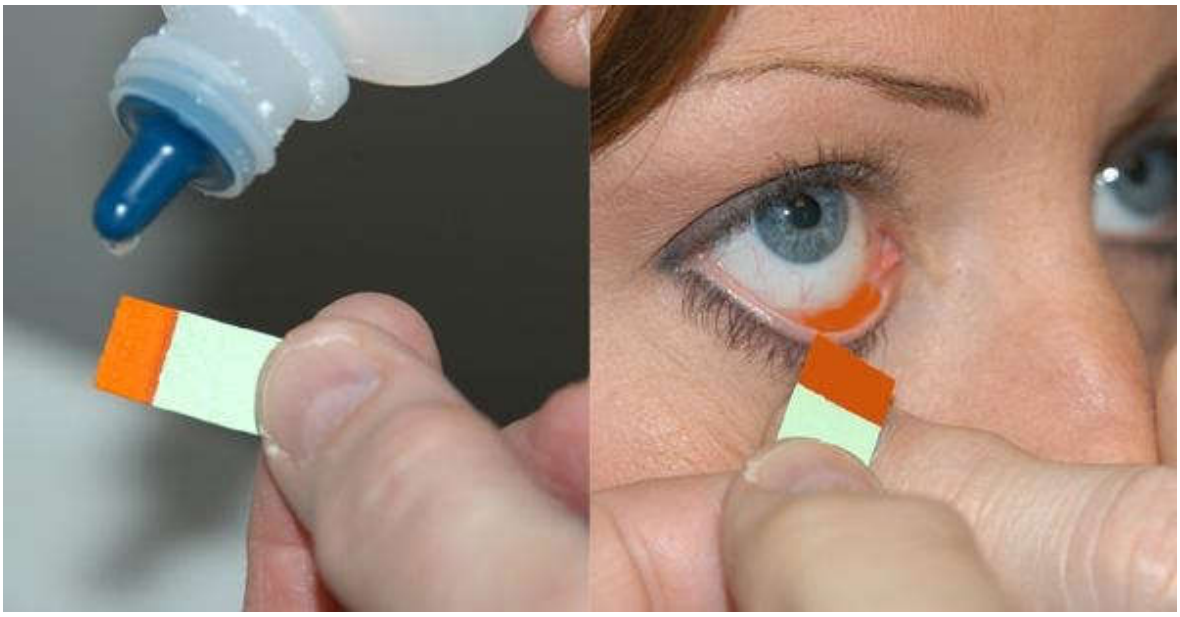
- Procedure should be done in a semidark room and patient can be asked to fix at a distant target through the other eye.

- The tension knob is kept at **1 g** to prevent vibrations and damage to the corneal epithelium. **0 graduation mark** of the prism is set at the white line on the prism holder.

  The slit beam should be **widest and brightest** with the filter is switched to **cobalt-blue filter** and the angle between the illumination and the microscope should be about **60°**to make slit beam shine through the tonometer head.

• Moistened ***1% sodium fluorescein dye*** strip is touched **on the inner fornix** and the patient should be asked to blink to spread the fluorescein-stained tear film over the cornea .

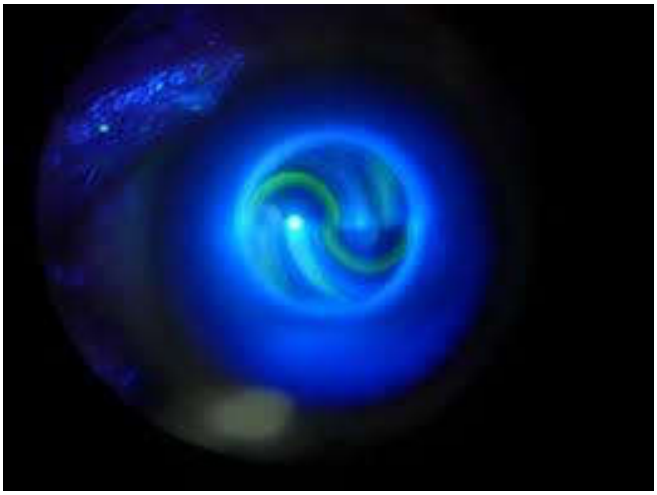
Ask patient to look slightly upwards not more than **15° above the horizontal** or examiner can hold the eyelids with forefinger and thumb resting on orbital rim to prevent the applanating unit from touching the lashes or lids

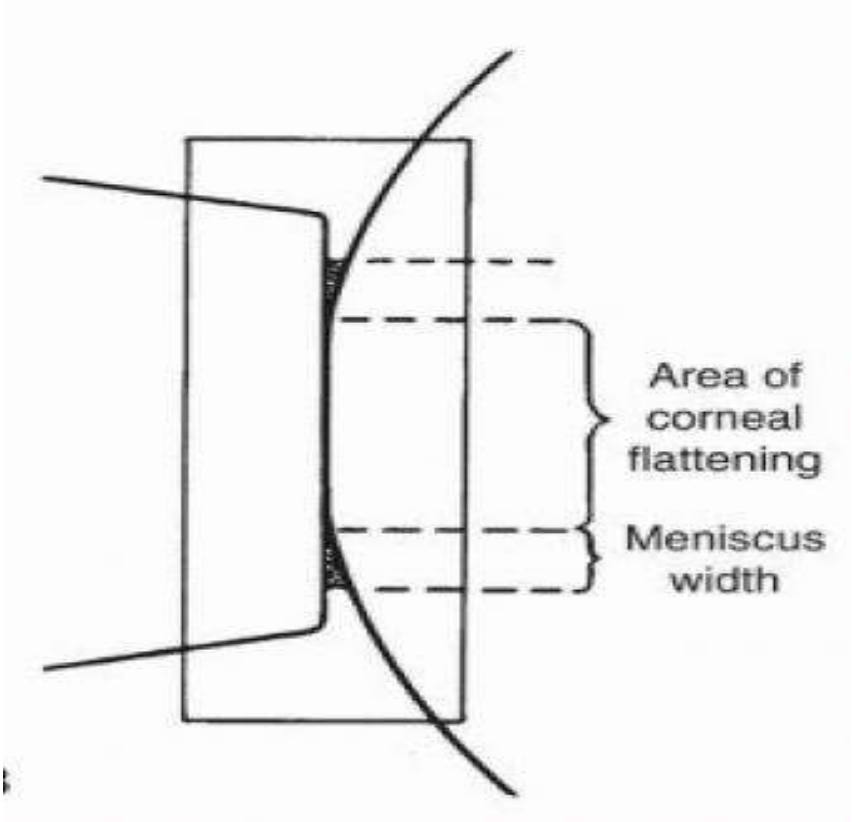


- Tonometer is kept perpendicular to the cornea and observed **monoocularly** through the biprism at **low magnification**. The instrument is advanced towards the patient until the tip of the prism gently touches the cornea and the semicircular mires are seen

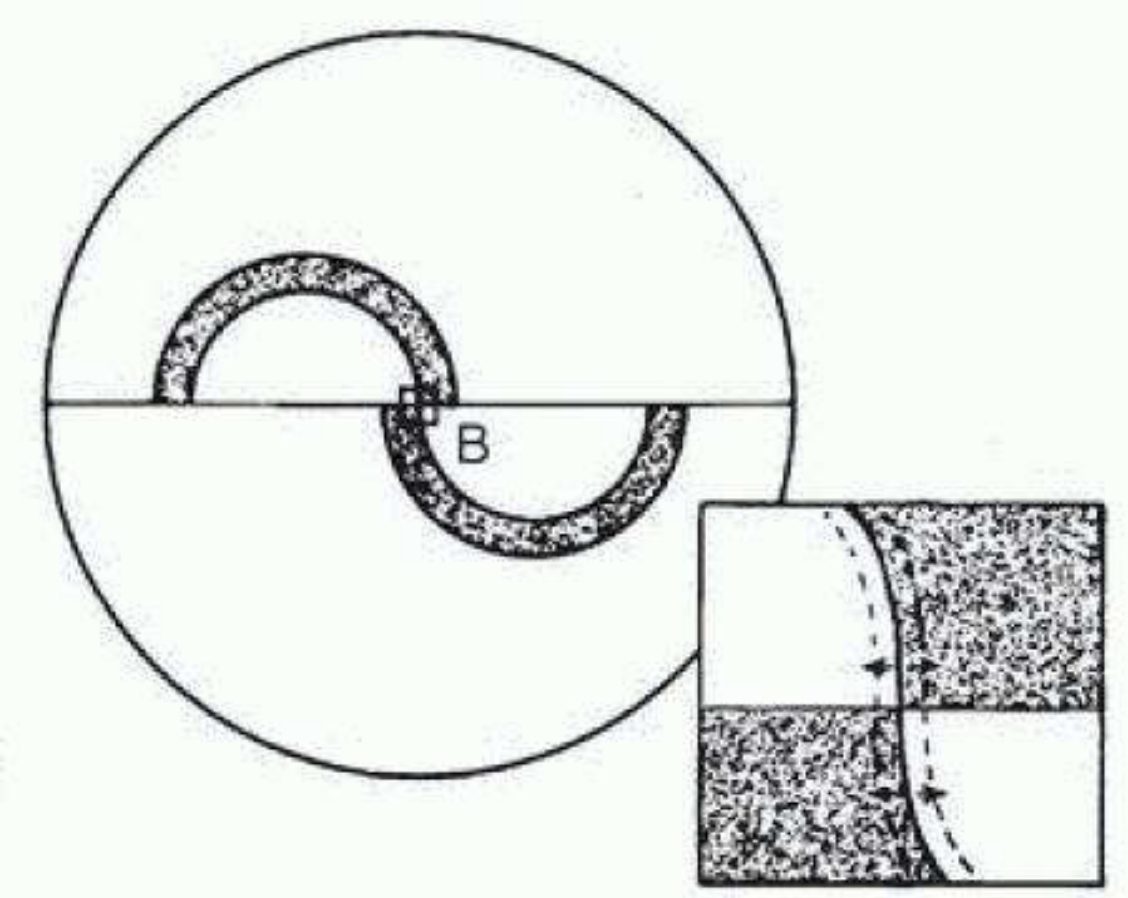
- Adjust until the two semicirclesare of equal size,optimum thickness and seen in the center of the field of view.

- The tension knob is rotated until the **inner borders** of the fluorescein rings meet each other at the **midpoint of their pulsations**





The reading measured in grams is multiplied by 10 to get the IOP in millimeters of mercury.



POSSIBLE ERRORS DURING MEASUREMENT:

1. Width of the fluorescein rings should be about one-tenth the diameter of the flattened area approximately **0.25–0.3 mm in thickness**

a. **too narrow rings- IOP is underestimated**

- patient should be asked to blink two or three times/ additional fluorescein should be added

b. **too wide - IOP is overestimated**

-the patient's eyelids should be blotted with a tissue/ the front surface of the prism should be dried with lint-free material

2. Corneal thickness –

a. Thick cornea →false high IOP

Ocular hypertension, due to additional tissue

b. Thin cornea →False low IOP

Normal pressure glaucoma, Following keratorefractive surgery

c. **Corneal edema – false low value** due to softening of the cornea although thickness is high

3. Corneal curvature –

a. an increase of approximately 1 mm Hg for every 3 diopters (D) of increase in corneal power.

b. Marked corneal astigmatism produces an elliptical area of corneal contact.

c. Rotate the biprism until the dividing line between the prisms is 45 degrees to the major axis of the ellipse, or an average may be taken of horizontal and vertical readings. Thus, minimizing the error.

d. With-the rule astigmatism→ False low IOP;

Against the-rule astigmatism→ False high IOP

4. An irregular cornea distorts the semicircles and interferes with the accuracy of the IOP estimates.

5. Prolonged contact with the cornea leads to corneal injury

6. A natural bias for even numbers may cause slight errors in readings.

7. Improper caliberation

FALSELY UNDERESTIMATE IOP	FALSELY OVERESTIMATE IOP
Thin cornea(cct <520micron)	Thick cornea(cct >550micron)
Inadequate fluorescein	Excessive fluorescein
Oedematous cornea	Steeper cornea
With the rule astigmatism	Against the rule astigmatism
1mmHG per 4D	1mmHG per 3D
Prolonged contact	Widening the lid fissure excessively
Repeat tonometry	Squeezing the eyelids/ pressing the globe

CALIBERATION OF THE INSTRUMENT:

GAT should be calibrated at least monthly.

To check caliberation, there is a weight bar provided along with the instrument. It consists of five circles – middle one for drum position 0, two immediately on either side for drum position 2 and two on either sides at the ends are for drum position 6

Drum setting with corresponding position in weight bar	Check position	Movement of feeler arm
0	0	Towards examiner
0	0.05	Towards patient
2	1.95	Towards examiner
2	2.05	Towards patient
6	5.9	Towards examiner
6	6.1	Towards patient

- If the GAT is not within 0.1 g (1 mmHg) of the correct calibration, the instrument should be repaired

- When the feeler arm is in the free movement zone, it should then move itself against the stop piece in the direction of the **patient**.

STERILIZATION

- tonometer tip soaked in diluted sodium hypochlorite, 3% H_2O_2 or 70% isopropyl alcohol for 5-15 mins

- wipe with alcohol, H_2O_2, povidone iodine or 1: 1000 merthiolate.

- rinse in running tap water for 10 min,

- wash with soap and water

- exposure to UV light

- cover the tip with a disposable film/ Disposable tonometer tips

*disposable tips/ shields have a smooth applanating surface but not 100% protective against prion disease.

➢ POSSIBLE INFECTIONS

Bacteria, viruses, and other serious infections such as epidemic keratoconjunctivitis, hepatitis B, Jacob-Kreutzfeld and also HIV.

Fluorescein preparation may be contaminated and may transfer Pseudomonas or Staphylococcus – can be prevented by using benoxinate, chlorobutanol, proparacaine or thiomersal

PRECAUTIONS

- Tip of the tonometer should be examined for a smooth applanating surface to prevent any corneal damage
- Care should be taken to rinse off the tipcompletely, as some alcohol-based ones, can be irritating /toxic to the epithelium corneal abrasion.
- Tonometer tip should be dry and wiped off before applying to the eye to prevent transfer of infection (e.g. Jacob-Kreutzfeld Virus) through the epithelial cells stuck on the tip.

CENTRAL CORNEAL THICKNESS

Normal corneal thickness is 0.7 to 0.9 mm at limbus and 0.49 mm and 0.56 mm at the centre. It also serves as a measure of corneal endothelial pump function and corneal rigidity. CCT acts as an independent risk factor for glaucoma and also to find out the actual IOP by applying correction factor . FACTORS AFFECTING CCT

1. Age – higher in children and decreases with increasing age
2. Gender –higher in male than females
3. Race – thinner in African Americans than white population
4. Refractive error – may be thinner in myopes
5. Diabetes – higher

USES

1. For diagnosis and monitoring the prognosis of certain corneal disorders
2. for correction of IOP readings measured by GAT according to the thickness of the cornea.
3. To decide the type of cataract surgery(ECCE instead of phacoemulsification)to be performed in cases with corneal disorders.
4. To monitor the status of the graft following keratoplasty

5. To monitor the thickness of the cornea in diseases such as keratoconus ,pellucid marginal degeneration.

ROLE OF CCT IN GLAUCOMA

- CCT is an important independent risk factor for glaucoma. According to the Ocular Hypertension Treatment Study, low CCT serves as an important risk factor for progression from ocular hypertension to early glaucoma.
- Higher or lower CCT values can affect the IOP measured by Goldmann Applanation Tonometry. Hence, actual IOP is obtained by applying correction factor according to variation in CCT
- Higher CCT values seen in ocular hypertension false high IOPs
- Lower CCT values in low tension glaucoma false low IOPs
- Lower CCT values also in POAG and pseudoexfoliation syndrome.

  No difference in PACG and pigmentary glaucoma patients

PACHYMETRY

Pachymetry is the method of measurement of corneal thickness. Greek words: *Pachos* - thick, *metry* - to measure. Measurement techniques :

1. Ultrasonic methods

   i. Conventional ultrasonic pachymetry

   ii. Ultrasound Biomicroscopy (UBM)

2. Optical methods

   i. Manual Optical Pachymetry

   ii. Specular Microscopy

   iii. Scanning Slit Technology

   iv. Optical Coherence Tomography(OCT)

   v. Optical Low Coherence Interferometry

   vi. Confocal Microscopy

   vii. Laser Doppler interferometry

3. Other Methods

   - Pentacam
   - Pachycam - Ocular response analyzer (ORA)

ULTRASONIC PACHYMETRY

Of these, **ultrasonic pachymetry is considered as the gold standard** and most commonly used since it is reproducible, faster, simpler and easy to use and also consistent and repeatable.

Principle: - Ultrasound energy is emitted from the probe tip acting as both the transmitter and receiver. Instruments functions by measuring the amount of time difference (transit time) between echoes of ultrasound pulse from the transducer and reflected signal from the front and back surface of the cornea.

Corneal thickness = (Transit time × Propagation velocity) / 2 Speed of sound in cornea-1640 m/sec is considered as standard

PARTS –

1. Probe handle –contains piezoelectric crystal . Vibrates at frequency of 10 to 20 MHz

2. Probe tip –smooth tipped with diameter not more than 2 mm.

3. Transducer – sends ultrasound rays and receives echoes from cornea through the probe

PROCEDURE :

- Patient is explained about the procedure and asked to sit erect.
- 0.5% Topical proparacaine eye drops is instilled into the conjunctival sac and patient is asked to close the eyes.
- Patient is instructed to look at the fixation light and not to blink his eyes during the procedure.
- The ultrasound probe is placed perpendicularly on the central part of cornea till the beep sound is heard and the reading is taken.
- 5 readings are taken and the average of the readings is taken as the central corneal thickness (CCT) in microns

PRECAUTIONS :

Probe tip should be smooth to prevent damage to the cornea

Probe tip should not be more than 2 mm to prevent decrease in accuracy due to wider probe and wider transducer beam

Probe should be perpendicular to the central cornea while measurement since oblique placement or lateral displacement of probe can lead to false high values.

ADVANTAGES

- Faster, simpler, easier, portable and doesn't require special training to handle
- No coupling medium required
- Minimal interobserver variation consistent, repeatable
- Can be used intraoperatively

DISADVANTAGES

Contact method - Cannot be done in cases of infection,

- Inaccurate if improper placement of probe
- Applanation force can decrease the thickness of the epithelium while measurement
- Corneal abnormalities such as edema may give inaccurate readings

INTRAOCULAR PRESSURE AND CENTRAL CORNEAL THICKNESS:

IOP is the only factor known to be amenable to treatment in glaucoma and glaucoma suspects.

Goldmann Applanation Tonometry(GAT) is the gold standard for clinical measurement of IOP. It is based on Imbert Fick's law, which assumes that cornea is a perfect flexible, dry, sphere which is infinitely thin. Therefore increase in the tissue in thicker cornea makes it less compliant and subsequently leading to overestimation of IOP and viceversa .

Ocular Hypertension Treatment Study (OHTS) group published that central corneal thickness (CCT) was an important independent risk factor for progression from ocular hypertension to early glaucoma(70% RISK).

In addition to this, the mathematical calculation for Goldmann applanation tonometry is based on a presumed average CCT of 550 μm, whereas the mean central corneal thickness in healthy human eyes is about 545 μm (micrometers).

Hence, the applanation tonometry readings are falsely high in thicker cornea(ocular hypertension, due to additional tissue) and falsely low in thinner corneas(normal tension glaucoma,following refractive surgery) respectively. So, the IOP values of applanation tonometry need to be adjusted according to variation in CCT from presumed average of 550 μm and a correction factor is always recommended to get the true IOP, to be considered for diagnosis and treatment.

According to a study by Ehlers and colleagues, the average error is 0.7 mm Hg per 10 μ of deviation from the mean of 550 μ, whereas another study, revealed a smaller error, of 0.19 mm Hg per 10 μ. Since the relationship between CCT and IOP is not linear, so even if the correction factor is applied, the correction of IOP over the extreme values of CCT becomes inaccurate and not reliable. Also, none of the correction factors, so far proposed, has been universally accepted as a standard formula.

PRESSURE-TO-CORNEA INDEX

So, to overcome this error in correction of IOP by various nonstandardized formulae, and also to integrate IOP and CCT as a single risk factor for glaucoma, Iliev*et al.,* introduced a new index called as **Pressure-To-Cornea Index(PCI).**

"PCI is the ratio between the highest recordable pretreatment IOP in mm Hg to the Central Corneal Thickness(CCT) in mm to the power of three".

$$PCI = \text{Pretreatment IOP (mm Hg)} \; CCT^{3} \; (mm)$$

In the study by Iliev et al, The Mean PCI in normal individuals, POAG, NTG and OHT were studied which were 92.0, 173.6, 129.1and 134.0 respectively. PCI had a significantly higher sensitivity of 80% and specificity of 90% when compared with each of the correction formulas such as Ehlers et. al , Doughty et. al, Shimmyo et. al. PCI in the normal individuals was in the range of 80 to 100 and a range of 120–140 may be considered as the upper limit of ''normality'' according to the study.

Uses:

To better differentiate glaucoma from non-glaucoma than each of the individual parameters alone.

As an indicator of risk for pressure related damage to the optic nerve due to glaucoma

To predict the susceptibility of the individual towards glaucomatous damage.

To set the target IOP for glaucoma treatment

To grade the severity of glaucoma.

In another study by Franco et al., it was found that PCI can also be used to grade the severity of glaucoma

PRESSURE-TO-CORNEA INDEX IN PRIMARY OPEN ANGLE GLAUCOMA

Primary open angle glaucoma is the most prevalent type of glaucoma with prevalence of 1 in 100 population in >40yrs of age. It has more aggressive clinical course with high IOP readings if not treated with medications at proper time .

Further, Corneal thickness in primary open angle glaucoma has been found to be variable, either thick or thin. So, CCT remains to be a highly variable factor in case of POAG eyes which in turn will affect the IOP in extreme CCT values.

So, the PCI can be used in such POAG eyes, where corrected IOP is not accurate.

PCI IN OCULAR HYPERTENSION AND NORMAL TENSION GLAUCOMA:

Also, in ocular hypertension group and normal tension glaucoma group, there is no linear relationship between IOP and CCT and applying correction factow was also found to be unreliable.

PRIMARY OPEN ANGLE GLAUCOMA

Chronic progressive optic neuropathy in adults in which there is a characteristic acquired atrophy of optic nerve & loss of retinal ganglion cells & their axons. This condition is associated with an open anterior chamber angle by gonio. Definition of POAG is not synonymously/ solely defined by presence of raised IOP.

EPIDEMIOLOGY:

Most common form of glaucoma in many countries & accounts for 60-70% cases in the united states. Worldwide over 2million develop this condition every year. Worldwide over 3 million are bilaterally blind because of POAG. The most recent US study estimated the overall prevelance of POAG as 1.9% above 40yrs, with blacks having three times the prevelance of whites.

RISK FACTORS:

1) IOP FLUCTUATION: increased IOP is one of the risk factor for glaucomatous damage in POAG. Even in patients with normal IOP said to have glaucomatous damage (NORMOTENSIVE GLAUCOMA) & patients with elevated IOP without evidence of glaucomatous damage (OCULAR HYPERTENSION). Hence IOP cannot be the only riskfactor.

2) TRANSCRIBROSAL PRESSURE DIFFERENTIALE: difference between CSF pressure & IOP across lamina cribrosa producing papilledema or glaucomatous cupping. CSF pressure decreases after 50 years of age.

3) Low BMI less than 18 is a increased risk factor for glaucoma. High BMI people tend to have high CSF pressure & hence maintains translaminar pressure gradient & so possess low risk for glaucoma. BMI is lesser in females.

4) AGE: POAG prevalence increases with age. Age is also a risk factor for conversion from ocular hypertension to open angle glaucoma.

5) GENDER: several studies have shown the prevalence of glaucoma to be more in males.

6) RACE: POAG is more prevalent in blacks. Disease seems to develop at an earlier age with rapid progression in black patients.

7) REFRACTIVE ERROR: myopia has been found to be associated with POAG.

8) CORNEAL THICKNESS: thin cornea is a risk factor for conversion from ocular hypertension to open angle glaucoma. In thin corneas IOP is underestimated. Thin corneas may be a marker for increased susceptibility of the optic nerve.

9) HEREDITY: 20-25% cases of POAG are hereditary. Risk of developing POAG in first degree relatives is 4-16%. Genes which are involved are GLC1A on chromosome 1, GLC1B on chr.2, MYOC gene(myocilin gene), OPTN gene (optineurin gene).

10) SYSTEMIC FACTORS: Diabetes affects the small blood vessels that supplies the optic nerve, thereby making it more susceptible to glaucamatous damage. Hypothyroidism, systemic vascular disease, sleep apnoea are associated with POAG.

PATHOPHYSIOLOGY:

REDUCED AQUEOUS HUMOUR OUTFLOW FACILITY, is the cause for raised IOP in POAG. It could be due to accumulation of foreign material in the trabecular meshwork, narrowing of trabecular pores, loss of endothelial cells in the trabecular meshwork resulting in loss of phagocytosis, decreased number of giant vacuoles which helps in moving fluid from trabecular meshwork to schlemms canal, narrowing of collector channels, closure of Schlem's canal.

↓

Results in elevated IOP

↓

Causes mechanical & vascular damage to the optic nerve head.

↓

Reduction in axoplasmic flow → decreased neurotropins & accumulation of metabolites, increased glutamate levels due to ischaemia

↓

Retinal ganglion cell death by apoptosis.

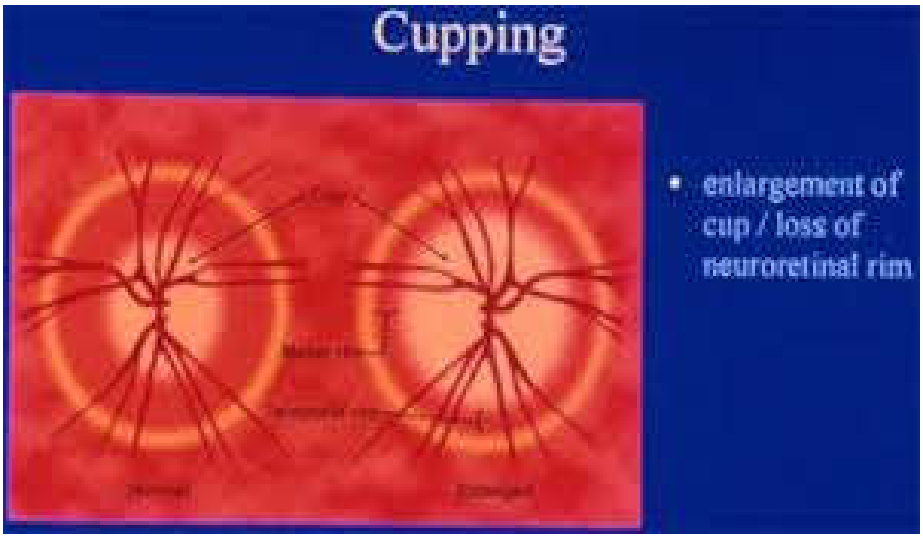
CLINICAL FEATURES:

SYMPTOMS: usually asymptomatic until late stages of the disease.

Occasionally patient can complain of coloured haloes due to corneal edema during sudden, severe elevation in IOP. Patient can notice a scotoma while doing monocular visual tasks.

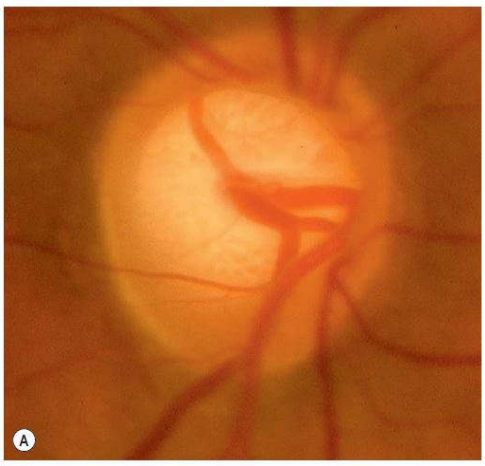
SIGNS:

1) VISUAL ACUITY: likely to be normal except in advanced glaucoma.
2) PUPILS: RAPD may be present when there is an asymmetrical cupping.
3) TONOMETRY: IOP may or may not be raised.
4) PACHYMETRY: central corneal thickness to be measured in order to get the corrected IOP.
5) GONIOSCOPY: done using goldmann 3 mirror gonio lens. Angles are open (>GRADE 2) by Shaffers grading. (Commonly used grading system is Shaffers grading.)
6) FUNDUS EXAMINATION:

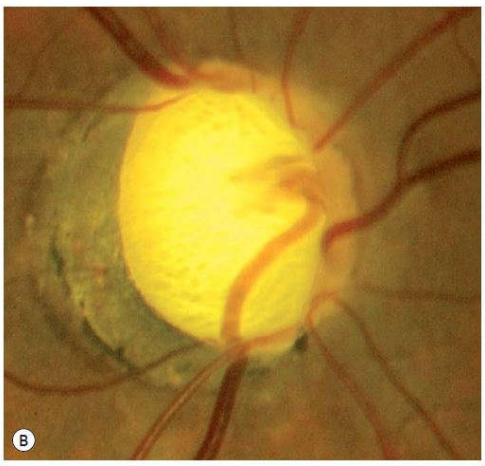


shows characteristic glaucomatous cupping with increased cup to disc ratio of > 0.5/ asymmetry of > 0.2 between both eyes.(normal c:d ratio is 0.3:1). Pathological cupping occurs due to an irreversible decrease in the number of nerve fibres, glial cells & blood vessels. With progressive cupping there occurs thinning of neuroretinal rim(space between the cup & disc margin). Thickness of nerve fibre bundle / follows ISNT rule (Inferior, superior, nasal and temporal).

Subtypes of glaucomatous damage includes following:



TYPE – 1



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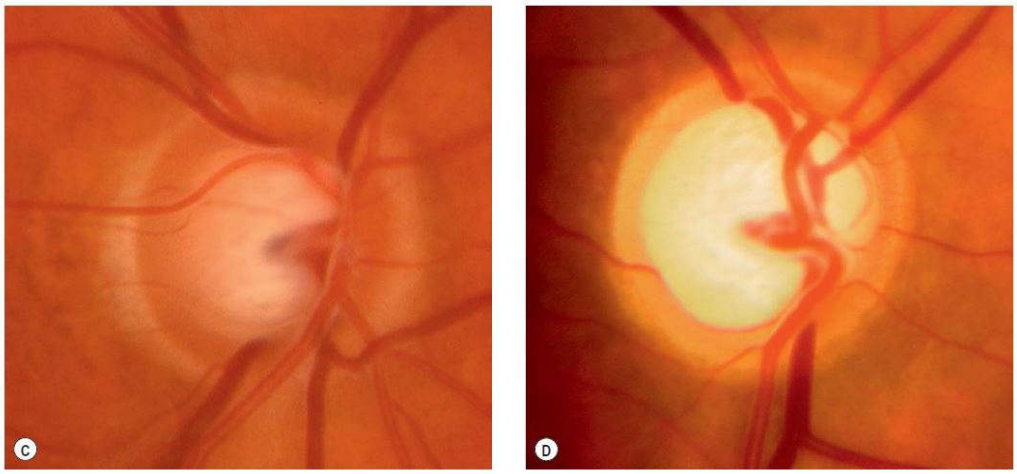


TYPE – 3 TYPE -

TYPE 1/ FOCAL ISCHAEMIC DISC: with focal superior/ inferior polar notching.

TYPE 2/ MYOPIC DISC WITH GLAUCOMA: tilted shallow disc with temporal crescent along with glaucomatous damage.

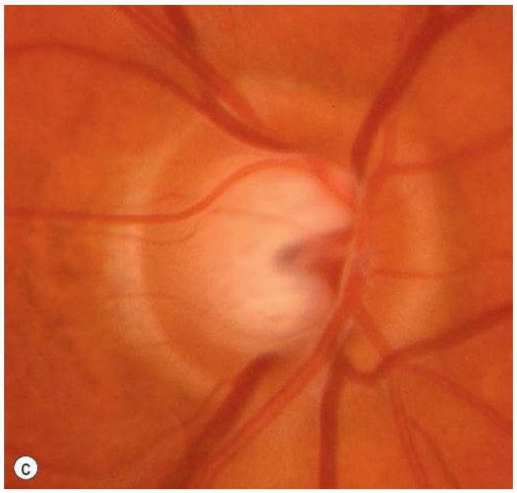
TYPE 3/ SENILE SCLEROTIC DISC: shallow, saucerised with a gently sloping NRR, variable peripapillary atrophy.

TYPE 4/ CONCENTRICALLY ENLARGING DISC: with uniform NRR thinning. IOP is significantly elevated.

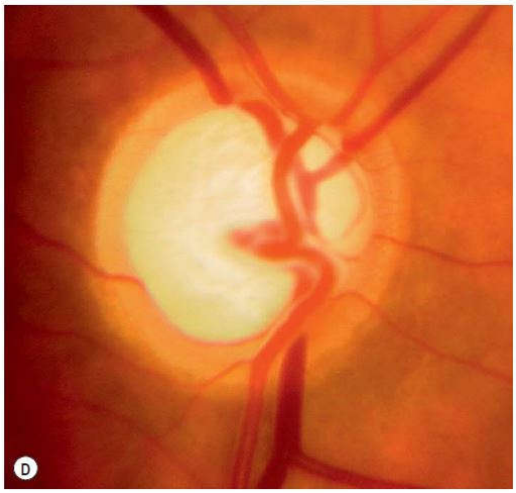
NON SPECIFIC SIGNS OF GLAUCOMATOUS DAMAGE:

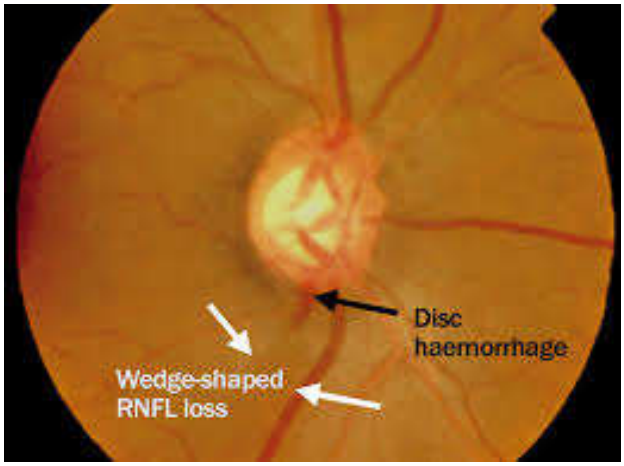
BARING OF CIRCUMLINEAR VESSELS: sign of early thinning of NRR. Characterised by a space between a superficial blood vessel that runs from superior/inferior aspect of the disc towards the macula & disc margin.

BARING OF CIRCUMLINEAR VESSELS



BAYONETTING





- BAYONETTING:

  kinking/ bending of the vessel over the edge of the cup.

- COLLATERALS BETWEEN TWO VEINS AT THE DISC.
- LAMINA DOT SIGN: occurs in advancing glaucoma. Grey dot like fenestrations in the lamina cribrosa becomes exposed as the NRR recedes. It may be seen in normal eyes.
- DISC HEMMORHAGES: extends from NRR onto the retina, most commonly in the inferotemporal quadrant. It is a risk factor for glaucoma & marker of inadequate control. Can occur even in healthy individuals, hypertensives & patients on antiplatelets.

- SHARPENED EDGE/RIM: sign of advancing damage. As the NRR is lost adjacent to the edge of the disc, disc margin contour assumes a sharper angle backwards.

- PERIPAPILLARY CHANGES: includes alpha/outer zone of peripapillary atrophy due to superficial retinal pigment epithelial changes. Beta/inner zone due to chorioretinal atrophy. These 2 zones are larger and more common in glaucoma.

- RETINAL NERVE FIBRE LAYER DEFECTS:

Subtle RNFL defects precede the development of detectable optic disc & field changes. Can be either a localised wedge shaped defect/ diffuse defect. Can be seen using red free light, confocal scanning laser tomography, OCT, GDX.

7)PERIMETRY : to detect visual field defects.

1) Paracentral, small relatively steep depressions constitute 70% of all early defects. Since the defects respect the distribution of retinal nerve fibre layer they terminate at the horizontal midline, defects above & below the horizontal midline are therefore not aligned with each other. Central/ paracentral scotoma may be most appropriately monitored using 10-2 humphrey perimetry pattern.

2) Nasal Ronne step represents a difference in sensitivity above & below the horizontal midline in the nasal field.

3) Arcuate shaped defects develop as a result of coalescence of paracentral scotomas. They typically develop between 10 & 20 degrees of fixation in areas that constitute downward, or more commonly upward extension from the blind spot around fixation (bjerrum area). With time they tend to elongate circumferentially along the distribution of arcuate nerve fibres (siedel scotoma) & may eventually connect with blind spot (arcuate scotoma) reaching to within 5 degree of fixation nasally.

4) Enlargement of scotomas due to damage to adjacent fibres.

5) Deepening of scotomas & development of fresh scotoma.

6) A ring scotoma develops when arcuate defects in upper & lower halves of visual field join. Misalignment between the two often preserves the nasal step.

7) End stage changes are characterised by a small island of central vision typically accompanied by a temporal island. The temporal island is usually extinguished before the central.

Neurological field defects can be differentiated from glaucomatous field defects as follows.

Glaucomatous field defects are relative, incongruous & follow horizontal meridian unlike neurological field defects which are absolute defect, mostly congruous and follows vertical meridian.

FIELD DEFECTS INTERPRETATION IN AUTOMATED PERIMETRY:

Before interpreting the field defects in AP, reliability indices should be checked as follows:

FIXATION LOSSES- includes steadiness of gaze during the test. They are detected by using gaze monitors. It should be less than 20%.

FALSE POSITIVE- detected when a stimulus is accompanied by sound. If the sound alone is presented & the patient still responds then a false positive is recorded. It should be less than 33%.

FALSE NEGATIVE- detected by presenting a stimulus much (9db) brighter than threshold at a location where threshold has already been determined. It should be less than 33%.

GLOBAL INDICES: they are used principally to monitor progression of glaucomatous damage.

1) Mean deviation: indicates overall sensitivity of the field. Derived from averaging the total deviation values, with central points given more weight. More than 10db indicates glaucomatous damage.

2) Pattern standard deviation: measure of focal loss or variability within the field taking into account any generalised depression in the hill of vision. Increased PSD is a more specific indicator of glaucomatous damage.

3) Short term fluctuation: indication of consistency of responses during a single test. Tends to increase in glaucoma & with ageing. More than or equal to 4db is unreliable.

4) Corrected pattern standard deviation: consists of PSD corrected for SF to produce a value for focal field abnormality corrected for intratest variability.

5) Probability values: represents the likelihood that an abnormal value of this level will occur in a normal subject. The lower the p value, the more likely the result is abnormal.

Global indices should always be taken into account, on an average annual deterioration in mean total deviation of just over 1 db can be expected in treated patients.

Anderson's criteria :

1. Glaucoma hemifield test (GHT) : Outside normal limits on atleast two consecutive occasions.
2. Three or more nonedge points in a location typical of glaucoma all of which are depressed at $p < 5\%$ and one of which is depressed at $p < 1\%$ level on two consecutive occasion.

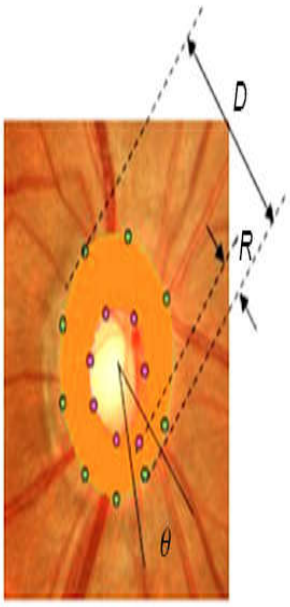
3. Corrected pattern standard deviation (CPSD) It takes into account the short term fluctuations, thereby highlighting the localized defects. It accounts for intra observer variations at p<5% level.

Criteria to grade glaucomatous field defects

Parameters	Early defects	Moderate defects	Severe defects
Mean deviation	< -6 dB	-6dB to -12 dB	> -12dB
Corrected pattern standard deviation	Depressed to the p< 5%	Depressed to the p< 5%	Depressed to the p< 5%
Pattern deviation plot i) Points depressed below p<5% ii) Points depressed below p<1%	<18 (25%) < 10	< 37 (50%) < 20	>37 (>50%) > 20
Glaucoma hemifield test	Outside normal limits	Outside normal limits	Outside normal limits
Sensitivity in central 5 degree	No point < 15dB	One hemifield may have point with sensitivity <15dB	Both hemifield have points with sensitivity < 15 dB

Disc Damage Likelihood Scale (DDLS)

		The thinnest width of the rim (Rim Disc Ratio)		
	Stage	Small disc <1.50 mm	Average size disc 1.50–2.00 mm	Large disc >2.00 mm
Normal	0a	0.5	0.4 or more	0.3 or more
	0b	0.4 up to 0.5	0.3–0.4	0.2–0.3
At Risk	1	0.3 up to 0.4	0.2–0.3	0.1–0.2
	2	0.2 up to 0.3	0.1–0.2	0.05–0.1
Glaucoma damage	3	0.1 up to 0.2	0.01–0.1	0.01–0.05
	4	0.01–0.1	No rim <45 degrees	No rim <45 degrees
	5	No rim <45 degrees	No rim 45–90 degrees	No rim 45–90 degrees
Glaucoma disability	6	No rim 45–90 degrees	No rim 91–180 degrees	No rim 91–180 degrees
	7	No rim >90 degrees	No rim >180 degrees	No rim >180 degrees



Rim disc ratio= $\frac{R}{D}$

CATEGORIES OF PRESENTATION:

Category 1- evidence available for both structural & functional damage- increased CD ratio 0.9, glaucoma field defect.

Category 2- evidence available only for advanced structural loss with unproved functional damage – eased CD ratio 0.9, but visual field could not be performed satisfactory by the subject.

Category 3- no clinical evidence available for both structural & functional damage- optic disc is not seen & visual fields are impossible. V/A less than 3/60, evidence of previous PI/ antiglaucoma surgery, from available old medical records confirming vision loss.

AAO GUIDELINES OF CLASSIFICATION OF PRIMARY OPEN ANGLE GLAUCOMA:

MILD POAG	:	upto 0.50 cupping . but, normal visual fields.
MODERATE POAG	:	0.50 to 0.75 cupping + one hemifield defect (but, not within 5 degrees of fixation point)
SEVERE POAG	:	0.50 to 0.75 cupping + both hemifield defect within 5 degrees of fixation point.

MANAGEMENT:

Primary aim is to prevent functional impairment of vision within the patients life time by slowing the rate of ganglion cell loss closer to that of the normal population (approx 5000/year).

Adequate instruction to be given to the patient regarding the nature of the disease, timing of medications & technique of application in order to ensure the compliance.

TREATMENT GOAL:

TO ACHIEVE THE TARGET PRESSURE- pressure level/ range of IOP below which further damage to the optic nerve head is unlikely. It is to be set on an individual basis, based on the baseline IOP, level of pre existing damage, rapidity with which the damage has occured, systemic associations. In general IOP is to be maintained below 18mmhg, fluctuations below 3 mm of Hg. Target IOP = INITIAL PRESSURE (1- INITIAL PRESSURE/100)Z+2, where Z is a constant. Based on the severity target pressure is set as follows:

MILD DISEASE: glaucomatous optic nerve head changes, normal visual fields. IOP to be reduced 20% from the baseline or below 18mmhg.

MODERATE DISEASE: visual field defects in one hemifield but not within 5 deg of fixation. IOP to be reduced 30% from the baseline or below 15mmhg.

SEVERE DISEASE: visual field defects in both hemifields/ within 5 deg of fixation. IOP to be reduced 50% from the baseline or below 13mmhg.

INDICATIONS FOR TREATMENT:

1) In presence of classical triad- field loss, optic nerve cupping, elevated IOP.

2) Progressive cupping without detectable field loss.

3) Development of visual field loss.

4) Episodes of corneal edema caused by raised IOP.

5) Vascular occlusion associated with raised IOP.

6) In case of asymetrical POAG better eye is usually agressively treated because it has 40% chance of developing field defects over a 5 year period.

7) Family history of POAG.

TREATMENT MODALITIES FOR POAG:

MEDICAL

LASER THERAPY.

SURGERY.

MEDICAL THERAPY:

1) PARASYMPATHOMIMETICS: pilocarpine 0.5%,1%,2%,4% eye drops qid as monotherapy. Causes 20-25% reduction in the IOP by contraction of the ciliary muscle, which increases the aqueous outflow through trabecular meshwork. Other miotics are carbachol 3% tid. Peak action seen in 3hrs & washout period is 1week. Side effects include miosis, browache, myopic shift of refraction, exacerbations of symptoms of cataract.

2) SYMPATHETIC DRUGS:

ALPHA 2 AGONISTS: causes 20-25% reduction in IOP by decreasing aqueous secretion & enhancing uveoscleral outflow. Because the **drug crosses the blood brain barrier** they should not be used in children. Side effects allergic conjunctivitis, acute granulomatous anterior uveitis, xerostomia, drowsiness, fatigue. Drugs are brimonidine 0.2% bd, apraclonidine 1%.

BETA BLOCKERS: causes 20-50% reduction in IOP by decreasing aqueous secretion. Useful in all types of glaucoma. Within few days of treatment there occurs short term escape & long term drift. In combination with prostaglandins an additional 20% reduction seen. Side effects include punctate epithelial erosions, reduced aqueous tear secretion, bradycardia, hypotension, bronchospasm, confusion, reduced HDL levels. Hence they are contraindicated in asthma, COPD patients where cardioselective beta blocker betaxolol 0.5% bd can be used. Eg; timolol 0.5% bd, levobunalol 0.5% bd, carteolol 1%, 2% bd, metipranolol 0.1%, 0.3% bd.

3) PROSTAGLANDIN ANALOGUES:

Causes 30-35% reduction in IOP by increasing the uveoscleral outflow. Side effects include conjunctival hyparemia, foreign body sensation, iris hyperpigmentation, eyelashes lengthening & hyper- pigmentation, cystoid macular edema, anterior uveitis, recurrence of herpetic keratitis. Eg; latanoprost 0.005% od, bimatoprost 0.03% od, travoprost 0.004% od, tafluprost 0.0015% od should not be given following cataract surgery.

4) CARBONIC ANHYDRASE INHIBITORS:

Causes 20-25% reduction in IOP by inhibiting aqueous secretion. Side effects include allergic blepharoconjuctivitis, transient bitter taste, precipitate corneal endothelial dysfunction, paraesthesia, malaise, gastritis, renal stones, Steven Johnson Syndrome, hypokalemia. Topical drugs are dorzolamide 2% tid, brinzolamide 1% bid/tid. Systemic drugs include Tab.Acetazolamide 250-1000mg daily in divided doses orally & 500mg powder vials for injection is also available, mathazolamide 50-100mg bid/tid.

5) HYPEROSMOTIC AGENTS:

They lower IOP by creating an osmotic gradient between blood & vitreous so that water is drawn out from the vitreous. Higher the gradient, greater the IOP reduction. To be effective they must be unable to penetrate blood aqueous barrier & hence of limited use in inflamatory glaucomas. Used when the IOP is acutely elevated prior to surgery as a temporary measure. Side effects include cardiovascular overload, urinary retention, headache, nausea. Drugs include 20% MANNITOL IV 1g/kg body weight or 5ml/kg over 30-60mins (duration of action 6hrs). Oral GLYCEROL 50% 1g/kg or 2ml/kg (duration of action is 3hrs), oral ISOSORBIDE 45% 1g/kg or 2ml/kg.

6) NEUROPROTECTIVE AGENTS:

NMDA receptor antagonist- memantine

Calcium channel blockers – nimodipine

Alpha 2 agonists- brimonidine

Vasodilators

Antioxidants

Citecholine.

IDEAL DRUG TO BE STARTED INITIALLY TO REDUCE THE IOP IS PROSTAGLANDIN ANALOGUES BUT SINCE THE DRUG IS EXPENSIVE & CANNOT BE AFFORDED BY ALL PATIENTS TIMOLOL HAS BEEN USED WIDELY AS THE FIRST OPTION OTHERWISE.

2) ARGON LASER TRABECULOPLASTY: IOP reduction is less than 30%.

INDICATIONS: intolerance of topical drugs including allergy, Failure of medical therapy as a less agressive treatment measure before surgery, Avoidance of polypharmacy more than two preparations, As a primary therapy in patients who are unwilling to comply with medical therapy.

LASER EFFECT:

Causes increased outflow facility by mechanical tightening of the trabecular meshwork to open the adjacent untreated trabecular spaces, and/or inducing cell division & migration of macrophages to clear the trabecular meshwork debris.

TECHNIQUE:

A drop of apraclonidine 1% is instilled to avert an early post laser IOP rise. Two drops of lignocaine applied. Gonio lens is inserted with mirror at 12'o clock position to visualise the inferior angle (easiest part to see). Argon laser beam (488-514nm) is aimed at the junction of pigmented & nonpigmented trabecular meshwork ensuring that the spot is round & has a clear edge. Laser settings are 50µm spot size, 0.1sec duration, 700mw power. If the reaction is inadequate power is increased by 200mw. 25burns are applied at regularly spaced intervals from one end of the mirror to other. Mirror is rotated 90deg clockwise & further 25 burns

applied hence making total of 50 burns over 180deg. Post laser topical predmet eye drops qid for a week along with antiglaucoma medications. Follow up done at 4-6weeks, if the IOP reduced significantly by 6 weeks, gradual withdrawal of topical drugs done. If IOP remains high another 180deg angle can be treated. But it is less likely to be beneficial & hence filteration surgery should be considered. Complications include peripheral anterior synaechiae, small hemorrhages, acute elevation of IOP, anterior uveitis, tenons cyst on subsequent filtration surgery. Initial success rate is 75-85%.

SELECTIVE LASER TRABECULOPLASTY:

New procedure which uses a 532 nm frequency doubled, Q switched Nd:YAG laser, which selectively targets melanin pigment in the trabecular meshwork cells, leaving non pigmented structures unscathed. Safer than ALT as there is no thermal tissue damage. 400µ, 3ms, 0.2-1.7 mw, 50-100 spots.

SURGERY:

INDICATIONS: failed maximally tolerated medical therapy when laser is likely to be inadequate or inappropriate, as a primary therapy in advanced disease requiring a very low target pressure may achieve a superior long term outcome from early surgery though risks must be carefully assessed on an individual basis.

TYPES OF FILTERING SURGERIES:

FULL THICKNESS - sclerectomy, trephination, thermal sclerostomy, laser sclerostomy, iridencleisis, goniopuncture.

PARTIAL THICKNESS: trabeculectomy.

NONPENETRATING: viscocanalostomy, canaloplasty, deep sclerectomy.

TRABECULECTOMY:

Introduced by late 1960 by cairns. Trabeculectomy lowers IOP by creating a fistula to allow aqueous outflow from the anterior chamber to the subtenon space. The fistula is protected or guarded by a superficial scleral flap. Goal is to produce complete healing of conjunctival wound with incomplete healing of scleral wound.

PROCEDURE: under stict aseptic precautions peribulbar block given. Superior rectus bridle suture applied. A limbal or fornix based flap is raised in the superior quadrant. Bleb is made at 12'o clock to reduce exposure & dysaesthesia.(In limbal based incision made 8-10mm from the limbus & chance of leakage is less. Fornix based flap provides easier exposure of the surgical site & reduces handling of the conjunctival flap & chance of leakage is more.) Tenons capsule is seperated. An outline of proposed superficial scleral flap is made with wet field cautery. Incisions are made along the cautery marks through two thirds of scleral thickness to create a trapdoor lamellar scleral flap. Flap may be triangular, trapezoidal/ rectangular (3*4mm). Flap is dissected forwards 1mm into the clear cornea. Anterior chamber is entered along the width of the trapdoor flap. 0.75-1mm of peripheral posterior cornea along with a block of deep sclera is removed using kellys descemets membrane punch. Peripheral iridectomy is done in order to prevent blockage of the internal opening. Superficial scleral flap is sutured at its posterior corners using 10-0 nylon so that it is lightly apposed to the underlying bed. Conjunctival flap is sutured with 8-0 vicryl. Inj. Dexamethasone 0.5cc given subconjuctivally. Post operatively Ab/st eye drops to be instilled 8times/day.

1. Novel pressure-to-cornea index in glaucoma

Br J Ophthalmol 2007;91:1364–1368. doi: 10.1136/bjo.2007.120980

Milko E Iliev, Alexander Meyenberg, Ernst Buerki, George Shafranov, M Bruce Shields

Background: Several conversion tables and formulas have been suggested to correct applanation intraocular pressure (IOP) for central corneal thickness (CCT). CCT is also thought to represent an independent glaucoma risk factor. In an attempt to integrate IOP and CCT into a unified risk factor and avoid uncertain correction for tonometric inaccuracy, a new pressure-to-cornea index (PCI) is proposed.

Methods: PCI (IOP/CCT3) was defined as the ratio between untreated IOP and CCT3 in mm (ultrasound pachymetry). PCI distribution in 220 normal controls, 53 patients with normal-tension glaucoma (NTG), 76 with ocular hypertension (OHT), and 89 with primary open-angle glaucoma (POAG) was investigated. PCI's ability to discriminate between glaucoma (NTG+POAG) and non-glaucoma (controls+OHT) was compared with that of three published formulae for correcting IOP for CCT. Receiver operating characteristic (ROC) curves were built.

Results: Mean PCI values were: Controls 92.0 (SD 24.8), NTG 129.1 (SD 25.8), OHT 134.0 (SD 26.5), POAG 173.6 (SD 40.9). To minimise IOP bias, eyes within the same 2 mm Hg range between 16 and 29 mm Hg (16–17, 18–19, etc) were separately compared: control and NTG eyes as well as OHT and POAG eyes differed significantly. PCI demonstrated a larger area under the ROC curve (AUC) and significantly higher sensitivity at fixed 80% and 90% specificities compared with each of the correction formulas; optimum PCI cutoff value

133.8.

Conclusions: A PCI range of 120–140 is proposed as the upper limit of ‘‘normality’’, 120 being the cut-off value for eyes with untreated pressures (21 mm Hg, 140 when untreated pressure >22 mm Hg. PCI may reflect individual susceptibility to a given IOP level, and thus represent a glaucoma risk factor. Longitudinal studies are needed to prove its prognostic value.

2. Correlation between the pressure-to-cornea index and both structural and functional measures of glaucoma

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Andrea M B V Franco, Niro Kasahara

Purpose: the pressure-to-cornea index (PCI) was proposed in order to integrate intraocular pressure and
central cornea thickness as a single-risk factor for glaucoma. The purpose of this study was to correlate
the PCI with a structural and two functional measures of glaucoma.

Setting: University Hospital in South America. Materials and Methods: Pressure-to-cornea index was calculated for 70 eyes of 36 subjects (glaucoma and suspects). Cup-to-disc (C/D) ratio, mean deviation (MD) and pattern standard deviation (PSD) as recorded by Humphrey automated perimetry (SITA 24-2) were correlated with PCI (Pearson’s correlation coefficient).

Results: Pearson's correlation coefficient between PCI and C/D was 0.329 (95% confidence interval [95% CI], 0.09–0.526; P = 0.006); between PCI and MD was − 0.356 MD (95% CI, −0.549 to − 0.126; P = 0.003); and between PCI and PSD was − 0.215 (95% CI, −0.433 to 0.025; P = 0.07).

Conclusion: In addition to serve as a single-risk factor, PCI can be used to stage glaucoma severity as well.

3. The Concurrent Pressure to Cornea Index Classifies Glaucoma Risk in Early Normal Tension Glaucoma

Ainur R. Anuar; Yingfeng Zheng; Huang Lei; Baskaran Mani; Carol Yim-Lui Cheung; Ching-Yu Cheng;Merwyn Chew; Jodhbir S. Mehta; Tien Yin Wong; Tin Aung

Purpose: To investigate the utility of an age-adjusted concurrent pressure to cornea index (CPCI) in discriminating between glaucoma and non-glaucoma.

Methods: CPCI was defined as the age-adjusted ratio between untreated intraocular pressure (IOP) and central corneal thickness^3 (CCT^3) in mm (IOP/CCT^3+4*age), measured within 2 hours of each other. In a population-based cross-sectional study, the distribution of CPCI in 294 normal controls (with normal visual fields), 124 with normal-tension glaucoma (NTG), 11 with ocular hypertension (OHT with normal visual fields), and 14 with primary open-angle glaucoma (POAG) was determined. CPCI's ability to discriminate between glaucoma (NTG+POAG) and non-glaucoma (normal+OHT) and between early stage NTG and normal was tested. In order to simulate the earlier stages of NTG, the better eye of NTG subjects (i.e. with smaller vertical cup:disc ratio, VCDR) was compared to the worst eye of controls (i.e. with larger VCDR) and only eyes with VCDR of less than 0.6 and IOP less than 22 mm Hg were

included. Area under the Receiver operating characteristic (ROC) curve was calculated to compare CPCI against IOP, CCT, and retinal nerve fibre layer thickness (RNFL) measurements (HRT II; Heidelberg Engineering).

Results: Mean (± SD) CPCI values were 303.13 (± 42.12) in controls, 357.17 (± 56.02) in NTG, 372.84 (± 46.62) in OHT, and 441.04 (± 62.98) in POAG. CPCI demonstrated larger AUC and significantly higher sensitivity at fixed 80% and 90% specificities (AUC=0.785, p<0.001) than other indices (IOP; AUC=0.534, p=0.257, CCT; AUC=0.577,p=0.01, mean RNFL; AUC=0.655, p<0.001). When eyes were controlled for IOP (<22 mmHg), VCDR (< 0.6) and subjected to conditions simulating early glaucoma, the performance of CPCI (AUC=0.849, p<0.001) improved further against IOP (AUC=0.557, p=0.251), CCT (AUC=0.615, p=0.020), and HRT II measured RNFL thickness (mean RNFL, AUC=0.557; p=0.298). Partitioning CPCI into three tertiles based on its mean and standard deviation ([T1] < 311, [T2]: 311- 359, [T3] > 359), compared to the lowest tertile (<311), the middle (311-359) and upper tertile (>359) of CPCI was associated with an odds ratio of 6.5 and 35.2 for NTG, respectively. A cut-off value of 310 as the upper limit of normality (or as having low risk for NTG), 311-359 as moderate risk, and 360 and above as high risk of NTG is recommended.

Conclusions: The CPCI has better discriminatory ability for glaucoma than IOP and CCT alone, and may be a useful summary indicator of glaucoma risk. Longitudinal studies are needed to prove its prognostic value.

AUTHOR'S CONCLUSION:

This review provides evidence that pressure –to-cornea index is a better indicator and independent risk factor for detecting glaucoma than CCT and IOP alone. They discussed that PCI may reflect individual susceptibility to a given IOP level and can be used to stage glaucoma severity as well.

PART TWO

AIMS AND OBJECTIVES:

- To integrate Intraocular Pressure(IOP) measured by Goldmann Applanation Tonometry (GAT) and Central Corneal Thickness(CCT) measured by ultrasonic pachymetry as a single risk factor in the form of Pressure-to-Cornea Index (PCI) for various IOP levels(ie, for POAG, NTG AND OCULAR HTN EYES).

- To determine the relationship of PCI in normal patients and POAG, NTG,OCULAR HTN patients.

- To find out the distribution of PCI in patients with POAG, NTG, OCULAR HTN and to find out whether PCI can be taken as a better predictor for glaucomatous damage in eyes with POAG, NTG, OCULAR HTN.

MATERIALS AND METHODS:

STUDY DESIGN:

Non randomized, comparative , cross-sectional study

Subjects for the study were chosen from patients attending ophthalmology department as outpatient as well as inpatient to the wards of our Govt.Rajaji Hospital, Madurai.

This study is to be conducted among 160 eyes of 80 patients above 40 years of age (of which 20 patients are normal subjects with no evidence of glaucoma, 20 patients with POAG, 20 patients with NTG, 20 patients with OCULAR HTN attending our department as outpatient as well as inpatient in the wards of our Govt. Rajaji Hospital, Madurai were included in this study.

Subjects shall be evaluated for entry into the study if they are 40 years of age or older. Subjects believed to fulfill eligibility criteria, and none of the exclusion criteria, will be invited to participate in the study.

STUDY PERIOD: 9 Months (December 2016 to august 2017)

SELECTION OF STUDY SUBJECTS:

A total of 160 eyes among patients with age of 40 years and above , attending as outpatient and in the wards of Department of Ophthalmology, Govt. Rajaji Hospital, Madurai who satisfy the inclusion criteria.

Subjects shall be evaluated for entry into the study if they are 40 years of age or older.

INCLUSION CRITERIA:

GROUP I -NORMAL SUBJECTS: comprising of eyes with following characteristics.

1. Age >= 40 years.
2. Baseline IOP <=21mmHg.
3. Open angles on gonioscopy.
4. Normal optic disc
5. No suspicion of any form of glaucoma
6. No other ophthalmic condition such as conjunctivitis, keratitis, uveitis, or any retinal pathology.

GROUP II - PRIMARY OPEN ANGLE GLAUCOMA PATIENTS : comprising of eyes with following characteristics.

1. Age >= 40 years.
2. Baseline IOP >21mmHg. Or history of IOP >21mmHg.
3. Open angles on gonioscopy
4. Glaucomatous optic disc –if any one or more of following features was present.

   a.increased cup size (cup :disc ratio >=0.4:1.0)

   b.neuroretinal rim thinning in any quadrant of the disc.

   c.notching of inferior or superior temporal area of optic nerve head.

   d.lamellar dot sign

   e.bayonetting sign

   f.hemorrhages on the disc

   g.total glaucomatous cupping.
5. Glaucomatous visual field defects

GROUP III – NORMAL TENSION GLAUCOMA PATIENTS : comprising of eyes with following characteristics

1. Age >= 40 years
2. Baseline IOP <21 mmHg.
3. Open angles on gonioscopy
4. Glaucomatous optic disc
5. Glaucomatous visual field defects

GROUP IV – OCULAR HYPERTENSION PATIENTS : comprising of eyes with following characteristics.

1. Age >=40 years
2. Baseline IOP >=21mmHg on more than two occasions
3. Open angles on gonioscopy
4. Normal optic disc

EXCLUSION CRITERIA

1. Patient age <40 years
2. Patients diagnosed to have secondary glaucoma
3. Evidence of any anterior segment pathology like conjunctivitis, keratitis or acute uveitis.
4. History of opaque ocular media (as in cataract, corneal opacities, vitreous opacities).
5. History of diabetes mellitus.
6. History of ocular surface disorders.
7. History of previous ocular surgery(cataract surgery/cornealsurgery)
8. History of chronic contact lens wear
9. History of previuos ocular trauma

10. Patients with occupation with exposure to higher temperatures such as glass blowers, furnace workers
11. Family history of glaucoma
12. Any H/O previous optic nerve disease
13. Any H/O intracranial disease or drugs affecting optic nerve head.
14. Any evidence of severe cardiac ,pulmonary , renal or liver disease.

Each case was examined in detail & observations were recorded on examination proforma.

ETHICAL COMMITTEE CLEARANCE

Obtained from the Ethical Committee of GRH Madurai.

FINANCIAL SUPPORT : nil

METHODOLOGY:

160 eyes of 8o patients 40 -70 years of age

20 normal subjects with no evidence of glaucoma,

20 patients with primary open angle glaucoma,

20 patients with ocular hypertension and

20 normal patients, were evaluated for PCI(pressure to corneal index).

All the patients were subjected to detailed clinical evaluation before being taken for the study. The evaluation will be done under following headings.

A) HISTORY:

1. age of onset
2. any precipitating factors
3. previous treatment (medical & surgical).

B) EXAMINATION:

1. Best corrected visual acuity.
2. Slit lamp examination.

C) DETAILED GLAUCOMA EXAMINATION:

1. Intra ocular pressure by Goldmann applanation tonometry
2. Central corneal thickness by ultrasonic pachymetry (ACCUPACH).
3. Angles by gonioscopy

4. Dilated fundus examination.

   a. media

   b. disc evaluation by +90D lens

   c. cup/disc ratio

   d. status of neuroretinal rim

   e. vascular pattern

   f. macula

5. visual field examination – by Humphrey's field analyser (white –on- white perimetry).

OBSERVATION AND ANALYSIS STATISTICAL METHOD:

The information collected regarding all the cases were recorded in a Master Chart.

Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software- range, frequencies, percentages, means, standard deviations, chi square,'t' value and 'p' values were calculated.

Student's 't' test was used to test the significance of difference between quantitative variables and Yate's and Fisher's chi square tests for qualitative variables.

A 'p' value of less than 0.005 is taken to denote significant relationship .

RESULTS AND ANALYSIS:

In our study, 160 eyes of 80 patients were included; 20 normal controls,20 with POAG, 20 with NTG, 20 with OHT were included. The demographic characteristics are given in table 1 and 2

TABLE 1: DISTRIBUTION BASED ON AGE AMONG FOUR GROUPS

Age in years	Normal	POAG	NTG	Ocular HTN	Total (%)
41 -50	10	1	4	5	20 (25%)
51 - 60	8	10	12	8	38(47.5%)
61 -70	2	9	4	7	22(27.5%)
Total	20	20	20	20	80
Mean	54.1	60.15	55.7	56.25	
SD	5.08	6.9	7.62	9.66	
p value	0.080 Not significant				

Table 1 shows age distribution among the patients in our study .The patients included in the study were between 40 to 70 years of age. Out of 80 patients, 20 patients (25%) were between 40 – 50 years of age ; 38 patients(47.5%) were between 51 – 60 yrs and 22 patients(27.5%) were between 61- 70 years.

The mean age of patients in POAG group , NTG group, OHT group were 60.15 , 55. 7, 56.25 years respectively.

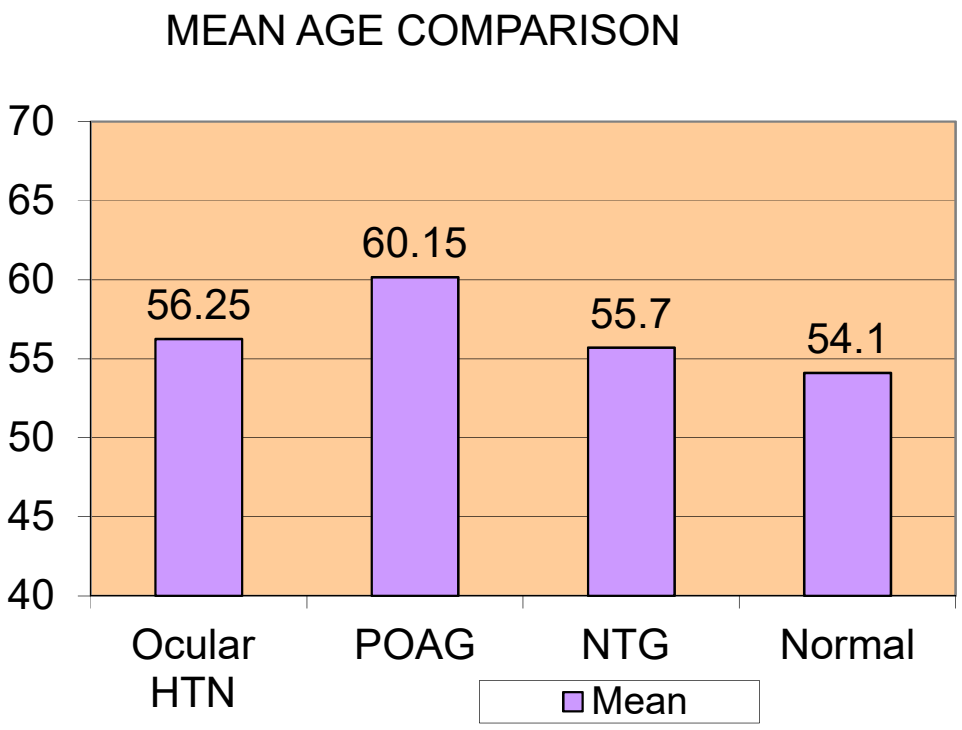


Table 2 shows the sex distribution among the four groups, wherein 53.75 % (43 patients) were males and 46.25% (37 patients) were females. There was equal sex distribution among four groups and was not statistically significant.

TABLE 2: DISTRIBUTION BASED ON SEX AMONG FOUR GROUPS

Sex	Normal	POAG	NTG	Ocular HTN	Total(%)
Male	12	11	9	11	43(53.75%)
Female	8	9	11	9	37(46.25%)
Total	20	20	20	20	80
p value	0.812 Not significant				

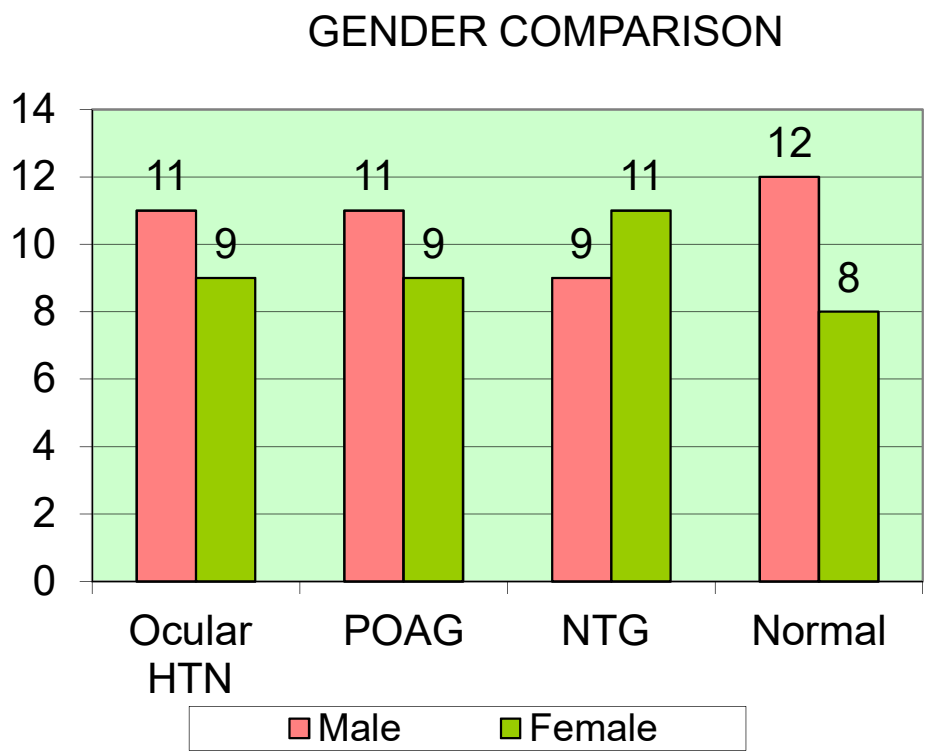


Table 3 shows the distribution of 160 eyes based on BCVA.

TABLE 3: DISTRIBUTION BASED ON BCVA AMONG FOUR GROUPS

BCVA	Ocular HTN	POAG	NTG	Normal
6 / 60	3	4	2	0
6 / 36	2	0	0	0
6 / 24	6	10	0	0
6 / 18	9	3	4	6
6 / 12	9	20	4	1
6/ 9	0	0	4	4
6 / 6	11	2	26	29
PL +	0	1	0	0
Total	40	40	40	40

Among the 40 eyes of OHT patients, 26 eyes had BCVA of 6/9 to 6/36 and 3 eyes had BCVA of 6/60.

Among 40 eyes of POAG patients, 33 eyes had BCVA of 6/9 to 6/36 and 4 eyes had BCVA of 6/60 and 1 eye had BCVA of perception of light.

Among 40 eyes of NTG patients, 12 eyes had BCVA of 6/9 to 6/18 and 2 eyes had BCVA of 6/60.

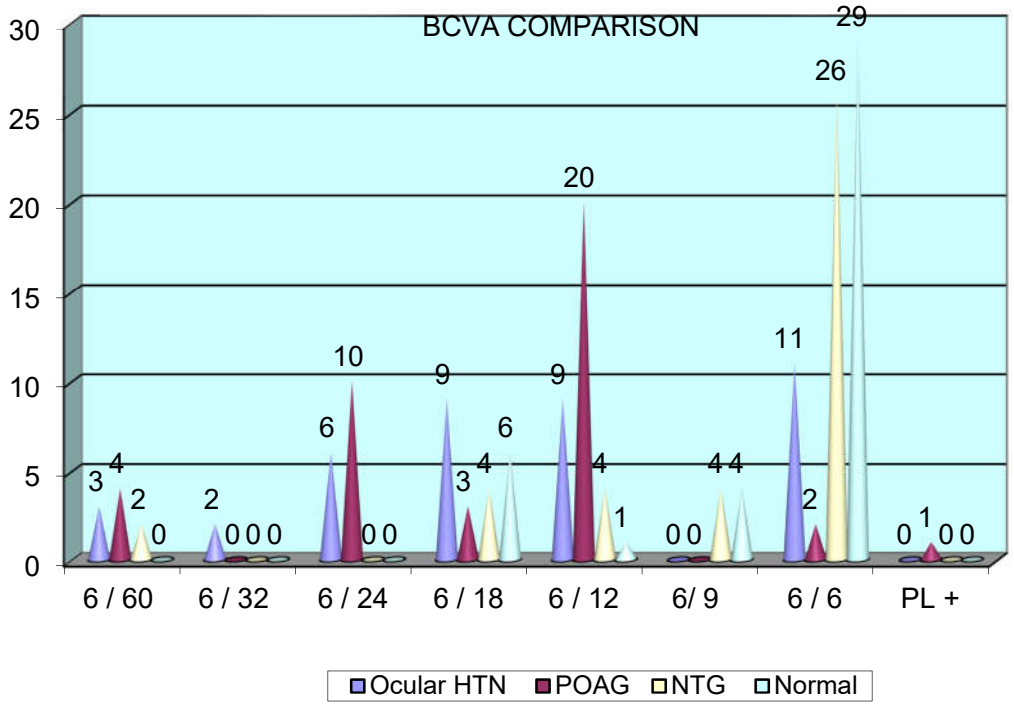
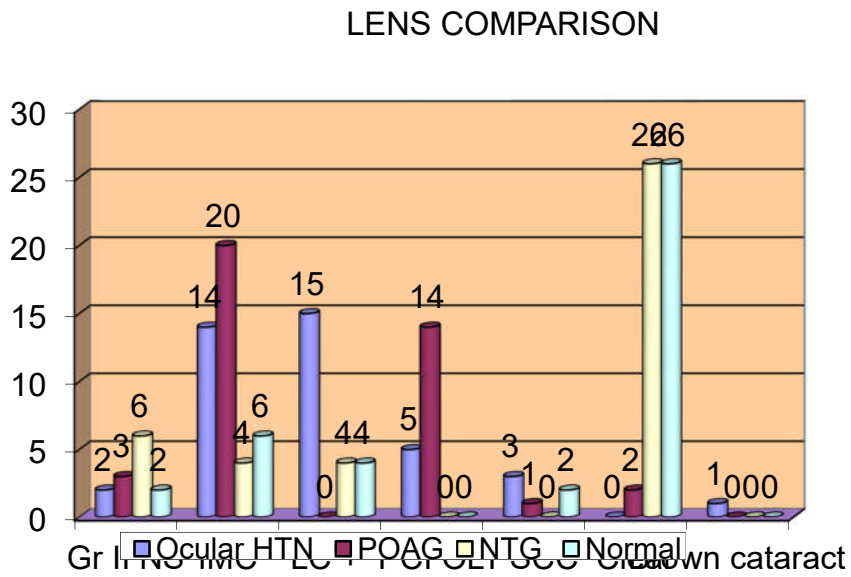


TABLE 4: DISTRIBUTION BASED ON LENS CHANGES AMONG FOUR GROUPS

Lens	Ocular HTN	POAG	NTG	Normal
Gr II NS	2	3	6	2
IMC	14	20	4	6
LC +	15	0	4	4
PCI OL	5	14	0	0
PSCC	3	1	0	2
Clear	0	2	26	26
Brown cataract	1	0	0	0
Total	40	40	40	40



In table 4 , all 40 eyes of OHT group had cataractous change, of which 5 eyes were operated for cataract with PCIOL implantation. Similarly, among POAG group , 38 had cataractous changes , of which 14 had underwent surgery

From table 5, the mean value of MIOP (mean intraocular pressure) in OHT, POAG, NTG , NORMAL groups were 26.8 , 19.55 ,16.1 and 16.05 mmHG respectively and this was statistically significant among the groups.

TABLE 5: DISTRIBUTION BASED ON MIOP AMONG FOUR GROUPS

MIOP	Ocular HTN	POAG	NTG	Normal
< 20	2	30	40	40
21 - 30	26	2	0	0
> 30	12	8	0	0
Total	40	40	40	40
Mean	26.8	19.55	16.1	16.05
SD	4.29	8.75	1.53	1.5
p value	< 0.001 Significant			

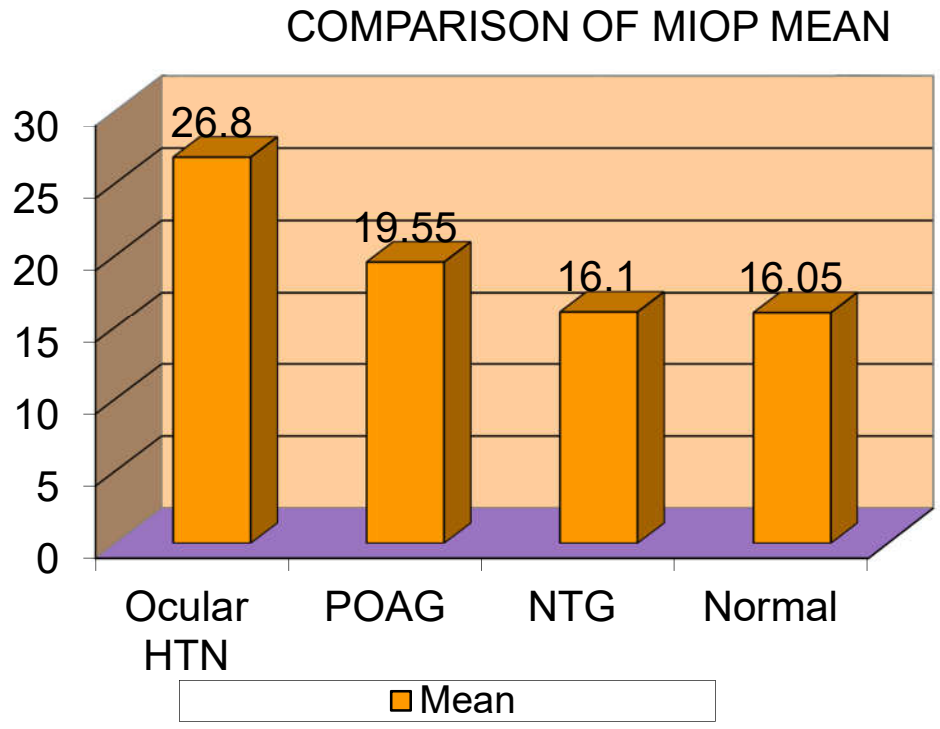
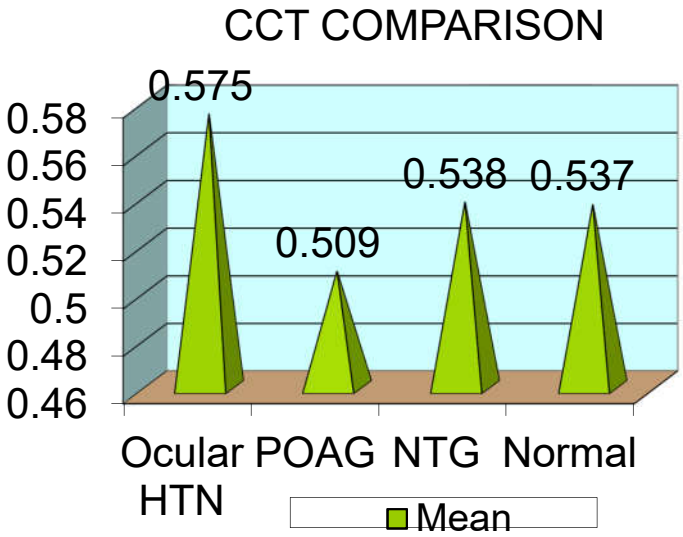


TABLE 6: DISTRIBUTION BASED ON CENTRAL CORNEAL THICKNESS AMONG FOUR GROUPS

CCT	Ocular HTN	POAG	NTG	Normal
< 0.5	1	15	7	7
0.5 - 0.6	25	25	33	33
> 0.6	14	0	0	0
Total	40	40	40	40
Mean	0.575	0.509	0.538	0.537
SD	0.047	0.036	0.032	0.029
p value	< 0.001 Significant			



The mean CCT value in the four groups viz., OHT, POAG, NTG and NORMAL , as shown in table 6 were 0.575 , 0.509 , 0.538 and 0.537 respectively with p value <0.001 showing a statistically significant difference among the four groups.

TABLE 7: DISTRIBUTION BASED ON TRUE IOP AMONG FOUR GROUPS

TIOP	Ocular HTN	POAG	NTG	Normal
< 20	5	20	35	33
20.1 - 30	25	10	4	7
> 30	10	10	1	0
Total	40	40	40	40
Mean	25.09	22.46	16.96	17
SD	5.09	9.32	2.42	2.7
p value	< 0.001 Significant			

From the above table , the mean value of TIOP (True intraocular pressure) in OHT, POAG, NTG,normal groups were 25.09 , 22.46 ,16.96 and 17.00 mmHG respectively and there was statistically significant difference among the groups.

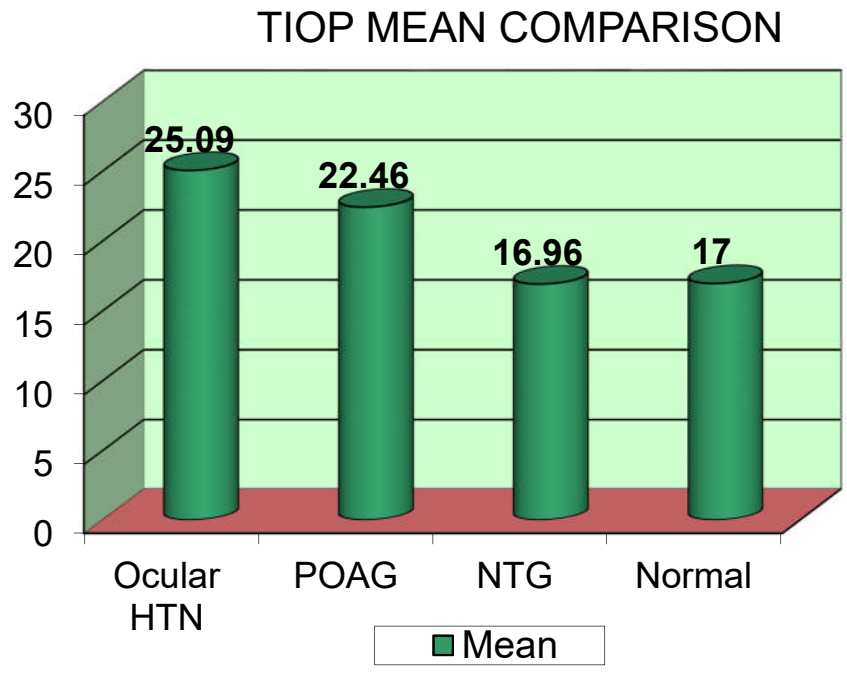


TABLE 8: DISTRIBUTION BASED ON PCI AMONG FOUR GROUPS

PCI	Ocular HTN	POAG	NTG	Normal
< 100	0	14	20	21
101 - 200	30	16	20	19
201 - 300	10	6	0	0
> 300	0	4	0	0
Total	40	40	40	40
Mean	147.00	154.41	105.42	106.03
SD	47.37	81.62	20.50	22.26
p value	< 0.001 Significant			

The PCI (pressure to cornea index) in 40 eyes of OHT group had a mean value of 147.00 with SD of 47.37 .The Mean and SD value of PCI for POAG group was 154.41and 81.62.

The NTG group had a MEAN PCI value of 105.42 with SD value of 20.50 while the normal group had a MEAN PCI of 106.03 and SD value of 22.26 . On statistical analysis, the p value was < 0.001 indicating significant differences among the four groups.

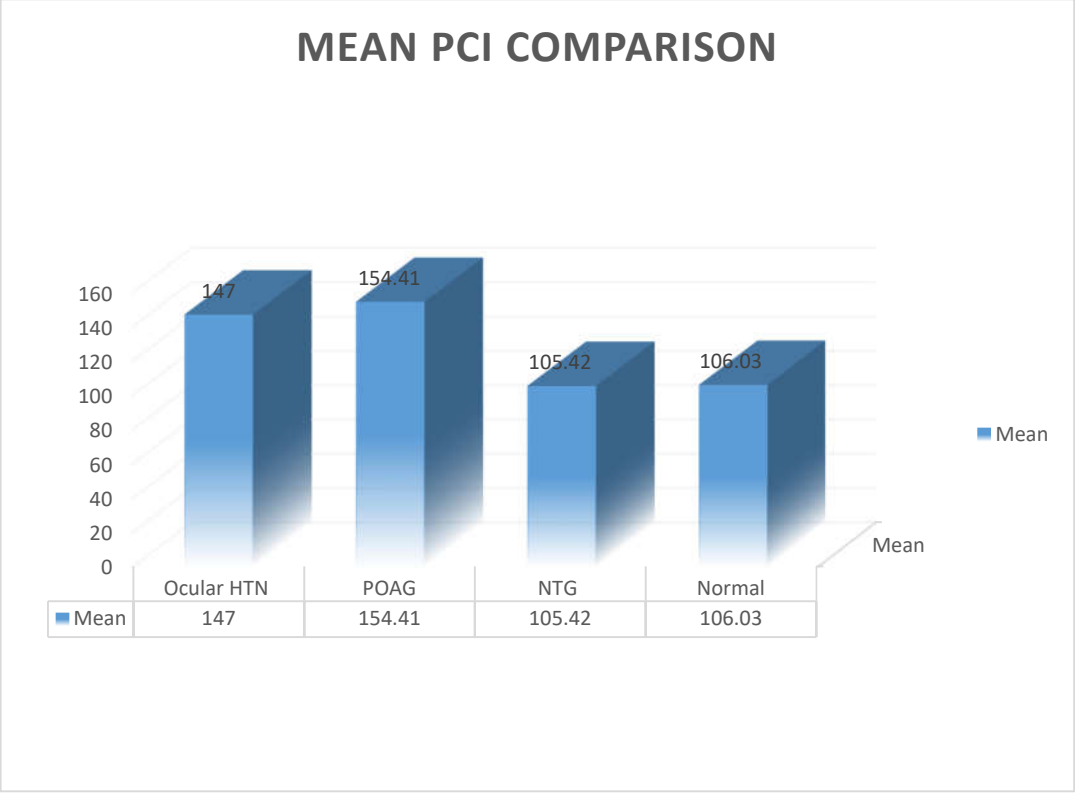


Table 9 and 10 showed field and disc changes among the four groups with field changes present in 31 eyes of POAG and 18 eyes of NTG group while disc changes were present in all eyes of POAG and almost all eyes of NTG group.

TABLE 9: DISTRIBUTION BASED ON FIELD CHANGES AMONG FOUR GROUPS

Field changes	Ocular HTN	POAG	NTG	Normal
Present	0	31	18	0
Absent	40	9	22	40
Total	40	40	40	40

TABLE 10: DISTRIBUTION BASED ON DISC CHANGES AMONG FOUR GROUPS

Disc changes	Ocular HTN	POAG	NTG	Normal
Present	0	40	38	0
Absent	40	0	2	40
Total	40	40	40	40

TABLE 11: CORRELATION BETWEEN MIOP, CCT,TIOP vs PCI AMONG OCULAR HYPERTENSION GROUP

Correleation in Ocular HTN	corr. Coeffi	Correlation
MIOP vs PCI	0.302	Low
CCT vs PCI	-0.884	Neg Good
TIOP vs PCI	0.831	Good

The above table shows the correlation between PCI value with MIOP, CCT and TIOP . As the PCI increases, CCT decreases and TIOP increases which was statistically significant.

TABLE 12: CORRELATION BETWEEN MIOP, CCT,TIOP vs PCI AMONG POAG GROUP

Correleation in POAG	corr. Coeffi	Correlation
MIOP vs PCI	0.754	Good
CCT vs PCI	-0.689	Neg Good
TIOP vs PCI	0.895	Good

Among the POAG group , there was a statistically significant correlation between PCI value with MIOP, and TIOP indicating , as the PCI increases, CCT decreases while MIOP and TIOP increases .

TABLE 13: CORRELATION BETWEEN MIOP, CCT,TIOP vs PCI AMONG NTG GROUP

Correleation in NTG	corr. Coeffi	Correlation
MIOP vs PCI	0.071	very Low
CCT vs PCI	-0.952	Neg Good
TIOP vs PCI	0.92	Good

Among the NTG group, comparing the PCI value with MIOP, CCT and TIOP shows with increase in PCI, the CCT decreases and TIOP increases and this correlation was statistically significant.

TABLE 14: CORRELATION BETWEEN MIOP, CCT,TIOP vs PCI AMONG NORMAL GROUP

Correleation in Normal	corr. Coeffi	Correlation
MIOP vs PCI	0.422	Moderate
CCT vs PCI	-0.955	Neg Good
TIOP vs PCI	0.948	Good

Among the Normal group, comparing the PCI value with MIOP, CCT and TIOP shows with increase in PCI , the CCT decreases and TIOP increases.

DISCUSSION:

- In our study, 160 eyes were considered with 40 eyes in each of the four groups-Ocular hypertension, Primary open angle glaucoma, Normal tension glaucoma and normal group.

- The demographic characteristics of the study group were as follows: age group from 40 to 70 years with maximum persons in 51 – 60 years of age (47.5%) with maximum mean age of 60.15 years in POAG group and minimum of 54.1 years in the normal group.

- There were 53.75 % of males and 46.25% of females totally.

- There was no statistically significant differences based on age and sex distribution among the four groups.

- Based on BCVA, 29 eyes of OHT group and 38 eyes of POAG group had impaired vision.

- The field and disc changes among the four groups were analysed, showing field changes in 31 eyes of POAG and 18 eyes of NTG group while disc changes were present in all eyes of POAG and almost all eyes of NTG group . This finding was in concordance with previous finding indicating absent field and disc changes in OHT group and presence of field and disc changes in POAG and NTG group.

- The mean value of MIOP (mean intraocular pressure) was highest in OHT group (26.8mmHG) followed by POAG group with the mean value of 19.55 mmHG. The mean values for the NTG and normal groups were 16.1 and 16.05 mmHG respectively .

- The mean CCT value was highest in OHT group (0.575 mm) and lowest in POAG group (0.509 mm) with 0.538 mm in NTG and normal groups and there was a highly significant statistical difference among the four groups. Thiis finding was similar to previous studies showing a thicker CCT in OHT and thinner CCT in POAG group. On comparing the mean of the CCT value among four groups showed a statistically significant difference among the groups.

- The TIOP was calculated as there is no linear correlation between MIOP and CCT. In our study , the highest mean value for TIOP was seen in OHT group which was similar to the previous studies. the POAG group had mean TIOP of 22.46mmHG.

- There was a statistically significant difference between MIOP and TIOP among all four groups which was similar to the findings of the study done by Ileiv et al .

- In this study, a new parameter , the pressure to cornea index was studied which is calculated based on pretreatment IOP and CCT. This PCI is novel parameter investigated by Iliev et al as a more precise glaucoma risk than either parameter alone.

- The mean PCI in 40 normal subjects was 106.03 whereas in the study by Iliev et al, most of the normal subjects had mean PCI of 80 to 100.

- The highest mean PCI was for POAG group with the value of 154.41 and SD was 81.62 , which was similar to the study done by Iliev et al , who investigated 89 eyes with POAG showing a mean value of 173.6.

- In our study, the mean PCI was 147 (SD of 47.37) for OHT group , which was slightly higher than the mean value of PCI according to Iliev et al , who studied 76 eyes of OHT (134.0).

- The NTG group in our study had a mean PCI value of 105.42 (SD of 20.50) which was slightly lower than the NTG group studied by Iliev et al (129.1)

- On analysing the mean PCI value among the four groups statistically, there was a significant difference observed.
- In our study, correlation between PCI with MIOP , CCT and TIOP were studied independently for each of the four groups which revealed the following findings:

    - OCULAR HYPERTENSION GROUP – PCI was significantly correlated with CCT and TIOP indicating positive correlation with TIOP and negative correlation with CCT. i.e, as the PCI increases, TIOP increases and CCT

decreases. However no significant correlation was observed with MIOP.

- PRIMARY OPEN ANGLE GLAUCOMA GROUP – There was a significant correlation between all three parameters with PCI , with positive correlation for TIOP and MIOP and a negative correalation with CCT.

- NORMAL TENSION GROUP- PCI was significantly correlated with CCT and TIOP indicating positive correlation with TIOP and negative correlation with CCT. i.e, as the PCI increases, TIOP increases and CCT decreases. However very low correlation was observed with MIOP.

- NORMAL GROUP – showed positive correlation with TIOP and negative correlation with CCT.

CONCLUSION

The following conclusions are made from our stusy.

1. Integrating TIOP and CCT into single risk factor in the form of pressure to cornea index (PCI) for four groups is better parameter than the independent parameters (TIOP and CCT) which is highly variable.

2. The relationship of PCI was significantly correlated among the four groups making it a more reliable parameter for various IOP levels.

3. Hence PCI may be taken as a independent glaucoma risk factor , as it can reflect changes in various levels of IOP and CCT.

LIMITATIONS

This is a pilot study with a small group of representative population in each group and hence the findings may not reflect the universal population.

Being a small cross sectional study, our study doesnot bring out the long term outcomes and also predictors of the variables.

Hence, large group sample and longitudinal studies are needed in the future.

PART THREE

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ABBREVIATIONS

MIOP – Mean Intraocular Pressure

TIOP – True Intraocular Pressure

CCT – Central Corneal Thickness

PCI – Pressure-to-Cornea Index

GAT – Goldmann Applanation Tonometry

POAG – Primary Open Angle Glaucoma

OHT – Ocular Hypertension

NTG - Normal Tension Glaucoma

PACG – Primary Angle Closure Glaucoma

BCVA- Best Corrected Visual Acuity

PROFORMA

Name: **Age:** **Sex:**

Glaucoma clinic No.:

Address: **Phone No:**

Presenting complaints:

Defective vision	**Duration**	**OD/OS/OU**
Pain		**OD/OS/OU**
Redness		**OD/OS/OU**
Coloured haloes		**OD/OS/OU**
Headache		
Frequent change of spectacles		
H/O DM/ HT/ BA/ IHD/ CKD/ PVD/ CVA	**Duration -**	
	Drugs -	
H/O Topical medication(ocular)		
H/O Trauma		
H/O Cataract/Glaucoma Surgery/LASER (PI)		
H/O Steroid oral/topical/nasal sprays		
Family history of glaucoma	**Yes/No**	

OD	Slit lamp examination	OS
	Lids	
	Conjunctiva	
	Cornea	
	Anterior chamber	
	Iris	
	Pupil	
	Lens	
	Visual acuity	

OD		OS
	IOP by NCT	
	IOP by AT	
	CCT	
	Gonioscopy	
	Fundus examination Media Disc CD Ratio Vessels N/B/PPA/LDS/ NRR/ BCLV Macula	
	Refraction by retinoscopy	
	Subjective vision	

	<u>Automated Perimetry (HFA)</u> **Reliabillity Indices** **MD** **PSD**	
	Colour vision	
	Pressure – Cornea Index **(IOP/ CCT3)**	

Diagnosis:

Treatment: MEDICAL -

SURGICAL -

Notes:

NORMAL GROUP

S.NO	NAME	AGE	SEX		BCVA	LENS	MIOP	CCT	TIOP	PCI	FIELD C	DISC CHANGES
				RE	6/6	NORMAL	18	0.536	19	116.89	ABSENT	ABSENT
1	PANIMADHA	42	F	LE	6/6	NORMAL	14	0.552	13.9	83.24	ABSENT	ABSENT
				RE	6/6	NORMAL	14	0.535	15.1	91.43	ABSENT	ABSENT
2	LAXMI	45	F	LE	6/6	NORMAL	15	0.512	17.7	111.76	ABSENT	ABSENT
				RE	6/18	PSCC	17	0.499	20.6	136.82	ABSENT	ABSENT
3	RAVIKUMAR	62	M	LE	6/18	GRIINS	18	0.534	19.1	118.21	ABSENT	ABSENT
				RE	6/6	NORMAL	15	0.532	16.3	99.62	ABSENT	ABSENT
4	RAJAN	48	M	LE	6/6	NORMAL	16	0.545	16.4	98.84	ABSENT	ABSENT
				RE	6/6	NORMAL	18	0.503	21.3	141.44	ABSENT	ABSENT
5	PARVATHI	58	F	LE	6/6	NORMAL	18	0.513	20.6	133.33	ABSENT	ABSENT
				RE	6/18	LC+	15	0.573	13.4	79.73	ABSENT	ABSENT
6	SANKARAN	58	M	LE	6/18	IMC	14	0.567	12.8	76.8	ABSENT	ABSENT
				RE	6/18	PSCC	15	0.552	14.9	89.18	ABSENT	ABSENT
7	VARADHAN	55	M	LE	6/9	GRINS	16	0.55	16	96.17	ABSENT	ABSENT
				RE	6/6	NORMAL	17	0.534	18.1	111.64	ABSENT	ABSENT
8	MAYIL	48	F	LE	6/6	NORMAL	16	0.586	13.5	79.51	ABSENT	ABSENT
				RE	6/18	IMC	17	0.498	20.6	137.65	ABSENT	ABSENT
9	KANNAN	57	M	LE	6/6	LC+	16	0.567	14.8	87.77	ABSENT	ABSENT
				RE	6/6	NORMAL	18	0.524	19.8	125.11	ABSENT	ABSENT
10	GANESAN	54	M	LE	6/9	IMC	14	0.553	13.8	82.79	ABSENT	ABSENT
				RE	6/12	IMC	17	0.544	17.4	105.6	ABSENT	ABSENT
11	KALIDASS	62	M	LE	6/6	NORMAL	14	0.551	13.9	83.69	ABSENT	ABSENT
				RE	6/6	NORMAL	13	0.521	15	91.92	ABSENT	ABSENT
12	RANI	49	F	LE	6/6	NORMAL	17	0.486	21.5	148.09	ABSENT	ABSENT
				RE	6/6	NORMAL	17	0.542	17.6	106.77	ABSENT	ABSENT
13	DEVI	51	F	LE	6/6	NORMAL	16	0.586	13.5	79.51	ABSENT	ABSENT
				RE	6/6	NORMAL	18	0.495	21.9	148.41	ABSENT	ABSENT
14	DHANDAPANI	49	M	LE	6/6	NORMAL	15	0.523	16.9	104.85	ABSENT	ABSENT
				RE	6/6	NORMAL	14	0.55	14	84.15	ABSENT	ABSENT
15	MURUGAN	48	M	LE	6/6	NORMAL	16	0.498	19.6	129.55	ABSENT	ABSENT
				RE	6/6	IMC	17	0.567	15.8	93.26	ABSENT	ABSENT
16	GANAPATHY	52	M	LE	6/6	IMC	17	0.568	15.7	92.77	ABSENT	ABSENT
				RE	6/6	NORMAL	15	0.489	19.3	128.28	ABSENT	ABSENT
17	THARANI	48	F	LE	6/6	NORMAL	18	0.552	17.9	107.02	ABSENT	ABSENT
				RE	6/9	LC+	16	0.552	15.9	95.13	ABSENT	ABSENT
18	NALLAN	48	M	LE	6/9	LC+	18	0.552	17.9	107.02	ABSENT	ABSENT
				RE	6/6	NORMAL	16	0.465	22	159.13	ABSENT	ABSENT
19	SIVAM	50	M	LE	6/6	NORMAL	18	0.567	16.8	98.75	ABSENT	ABSENT
				RE	6/6	NORMAL	15	0.551	14.9	89.67	ABSENT	ABSENT
20	DEVI	52	F	LE	6/6	NORMAL	14	0.538	14.8	89.9	ABSENT	ABSENT

NORMAL TENSION GLAUCOMA

S.NO	NAME	AGE	SEX		BCVA	LENS	MIOP	CCT	TIOP	PCI	FIELD C	DISC CH
1	POTHUMANI	54	F	RE	6/6	CLEAR	18	0.565	17	99.8	ABSENT	PRESENT
				LE	6/6	CLEAR	18	0.591	15.1	87.2	ABSENT	PRESENT
2	PALTHAI	60	F	RE	6/12	GR II NS	14	0.48	18.9	126.59	PRESENT	PRESENT
				LE	6/18	GR II NS	14	0.509	16.9	106.16	PRESENT	PRESENT
3	SHANTHI	55	F	RE	6/12	IMC	16	0.507	19	122.77	PRESENT	PRESENT
				LE	6/9	IMC	16	0.508	18.9	122.05	PRESENT	PRESENT
4	RAJA	42	M	RE	6/9	LC+	16	0.559	15.4	91.6	ABSENT	ABSENT
				LE	6/18	LC+	20	0.575	18.3	105.2	ABSENT	ABSENT
5	CHIDHAMBARAM	60	M	RE	6/60	GRINS	16	0.517	18.3	115.78	PRESENT	PRESENT
				LE	6/60	GRINS	16	0.519	18.2	114.45	PRESENT	PRESENT
6	KANAGAMARY	48	F	RE	6/6	CLEAR	14	0.567	12.8	76.8	PRESENT	PRESENT
				LE	6/6	CLEAR	15	0.552	14.9	89.18	PRESENT	PRESENT
7	AMMUTHAI	59	F	RE	6/6	CLEAR	16	0.55	16	96.17	PRESENT	PRESENT
				LE	6/6	CLEAR	17	0.534	18.1	111.64	ABSENT	PRESENT
8	KARUPAIAH	69	M	RE	6/6	CLEAR	16	0.586	13.5	79.51	ABSENT	PRESENT
				LE	6/6	CLEAR	17	0.498	20.6	137.65	ABSENT	PRESENT
9	JEYAKODI	68	F	RE	6/6	CLEAR	16	0.567	14.8	87.77	ABSENT	PRESENT
				LE	6/6	CLEAR	18	0.524	19.8	125.11	ABSENT	PRESENT
10	MADAN	70	M	RE	6/6	CLEAR	14	0.553	13.8	82.79	ABSENT	PRESENT
				LE	6/6	CLEAR	17	0.544	17.4	105.6	ABSENT	PRESENT
11	HARIHARAN	54	M	RE	6/6	CLEAR	14	0.551	13.9	83.69	ABSENT	PRESENT
				LE	6/6	CLEAR	13	0.521	15	91.92	ABSENT	PRESENT
12	KESAVAN	52	M	RE	6/12	GR II NS	17	0.486	21.5	148.09	ABSENT	PRESENT
				LE	6/18	GR II NS	17	0.542	17.6	106.77	ABSENT	PRESENT
13	KALIDASS	65	M	RE	6/12	IMC	16	0.586	13.5	79.51	PRESENT	PRESENT
				LE	6/9	IMC	18	0.495	21.9	148.41	PRESENT	PRESENT
14	FATHIMA	54	F	RE	6/9	LC+	15	0.523	16.9	104.85	PRESENT	PRESENT
				LE	6/18	LC+	14	0.55	14	84.15	PRESENT	PRESENT
15	AMBALAM	52	M	RE	6/6	CLEAR	16	0.498	19.6	129.55	ABSENT	PRESENT
				LE	6/6	CLEAR	17	0.567	15.8	93.26	PRESENT	PRESENT
16	DEVI	51	F	RE	6/6	CLEAR	17	0.568	15.7	92.77	ABSENT	PRESENT
				LE	6/6	CLEAR	15	0.489	19.3	128.28	ABSENT	PRESENT
17	DHANDAPANI	49	M	RE	6/6	CLEAR	18	0.552	17.9	107.02	PRESENT	PRESENT
				LE	6/6	CLEAR	16	0.552	15.9	95.13	PRESENT	PRESENT
18	MURUGAN	48	M	RE	6/6	CLEAR	18	0.552	17.9	107.02	ABSENT	PRESENT
				LE	6/6	CLEAR	16	0.465	22	159.13	ABSENT	PRESENT
19	GANAPATHY	52	M	RE	6/6	CLEAR	18	0.567	16.8	98.75	ABSENT	PRESENT
				LE	6/6	CLEAR	15	0.551	14.9	89.67	ABSENT	PRESENT
20	KAVERI	52	F	RE	6/6	CLEAR	14	0.538	14.8	89.9	PRESENT	PRESENT
				LE	6/6	CLEAR	16	0.552	15.9	95.13	PRESENT	PRESENT

PRIMARY OPEN ANGLE GLAUCOMA

S.NO	NAME	AGE	SEX		BCVA	LENS	MIOP	CCT	TIOP	PCI	FIELD CHANGES	DISC CHANGES
1	Pandiyammal	41	f	RE	6/12	PSCC	17	0.527	18.6	116.15	PRESENT	PRESENT
				LE	6/12	PCIOL	17	0.479	22	154.68	PRESENT	PRESENT
2	CHELLA DURAI	65	M	RE	6/18	IMC	20	0.537	20.9	129.15	PRESENT	PRESENT
				LE	6/24	IMC	20	0.539	20.8	127.72	PRESENT	PRESENT
3	RAMUTHAI	65	F	RE	6/12	PCIOL	12	0.479	17	109.19	PRESENT	PRESENT
				LE	6/12	PCIOL	12	0.51	14.8	90.46	PRESENT	PRESENT
4	BALU	60	M	RE	6/12	PCIOL	18	0.431	26.3	224.82	PRESENT	PRESENT
				LE	6/12	PCIOL	12	0.413	21.6	170.35	PRESENT	PRESENT
5	DHANABAL	60	M	RE	6/24	GR II NS	38	0.473	43.4	359.09	PRESENT	PRESENT
				LE	6/24	IMC	32	0.476	37.2	296.71	PRESENT	PRESENT
6	KATCHAMMAL	60	F	RE	6/12	PCIOL	36	0.448	43.1	400.38	PRESENT	PRESENT
				LE	6/24	GR III NS	40	0.509	42.9	303.32	PRESENT	PRESENT
7	MUNIYANDI	60	M	RE	6/12	PCIOL	16	0.489	20.3	136.83	PRESENT	PRESENT
				LE	6/60	GR III NS	14	0.487	18.4	121.21	PRESENT	PRESENT
8	AMMUTHAI	59	F	RE	6/12	PCIOL	12	0.512	14.7	89.41	PRESENT	PRESENT
				LE	6/12	PCIOL	11	0.558	10.4	63.31	PRESENT	PRESENT
9	KARUPAIAH	69		RE	6/12	PCIOL	32	0.533	33.2	211.33	PRESENT	PRESENT
				LE	6/24	IMC	32	0.527	33.6	218.63	PRESENT	PRESENT
10	JEYAKODI	68	F	RE	6/12	PCIOL	17	0.493	21	141.88	PRESENT	PRESENT
				LE	6/12	PCIOL	17	0.472	22.5	161.67	PRESENT	PRESENT
11	MADAN	70	M	RE	6/24	IMC	14	0.518	16.2	100.73	ABSENT	PRESENT
				LE	6/60	IMC	12	0.482	16.8	107.16	ABSENT	PRESENT
12	NAGAMMAL	61	F	RE	6/24	IMC	17	0.558	16.4	97.85	ABSENT	PRESENT
				LE	6/12	IMC	17	0.533	18.2	112.27	ABSENT	PRESENT
13	RATHINAPANDI	54	M	RE	6/6	CLEAR	14	0.542	14.6	87.93	ABSENT	PRESENT
				LE	6/6	CLEAR	14	0.595	10.9	66.46	PRESENT	PRESENT
14	DURAIPANDI	65	M	RE	6/60	IMC	16	0.551	15.9	95.65	ABSENT	PRESENT
				LE	6/24	IMC	28	0.514	30.5	206.19	ABSENT	PRESENT
15	RATHINAM	54	M	RE	6/12	PCOL	12	0.521	14	84.85	ABSENT	PRESENT
				LE	6/12	PCIOL	14	0.519	16.2	100.14	ABSENT	PRESENT
16	RAJENDRAN	67	M	RE	PL+	IMC	38	0.5	41.5	304	PRESENT	PRESENT
				LE	6/24	IMC	18	0.488	22.3	154.89	PRESENT	PRESENT
17	PANDI	54	M	RE	6/12	IMC	12	0.521	14	84.85	PRESENT	PRESENT
				LE	6/18	IMC	14	0.519	16.2	100.14	PRESENT	PRESENT
18	CHANDRAN	54	M	RE	6/24	IMC	28	0.517	30.3	202.62	PRESENT	PRESENT
				LE	6/60	IMC	32	0.558	31.4	184.18	PRESENT	PRESENT
19	MALLIKA	55	F	RE	6/12	IMC	12	0.512	14.7	89.41	PRESENT	PRESENT
				LE	6/18	IMC	14	0.487	18.4	121.21	PRESENT	PRESENT
20	RAJAMMAL	62	F	RE	6/12	IMC	14	0.542	14.6	87.93	PRESENT	PRESENT
				LE	6/12	IMC	17	0.472	22.5	161.67	PRESENT	PRESENT

OCULAR HYPERTENSION GROUP

S.NO	NAME	AGE	SEX		BCVA	LENS	MIOP	CCT	TIOP	PCI	FIELD CHANGE	DISC CHANGE
				RE	6/6	LC+	32	0.636	26	124.39	ABSENT	ABSENT
1	SYED	32	m	LE	6/6	LC+	26	0.622	21	108.04	ABSENT	ABSENT
				RE	6/6	LC+	30	0.59	27.2	146.07	ABSENT	ABSENT
2	PALANI	50	M	LE	6/6	LC+	22	0.601	18.4	101.34	ABSENT	ABSENT
				RE	6/12	PCIOL	18	0.541	18.6	113.68	ABSENT	ABSENT
3	PETCHI	50	F	LE	6/6	PCIOL	18	0.536	19	116.89	ABSENT	ABSENT
				RE	6/6	LC+	32	0.646	25.3	118.7	ABSENT	ABSENT
4	PULIYAMMAL	47	F	LE	6/6	LC+	26	0.627	20.6	105.48	ABSENT	ABSENT
				RE	6/18	BROWN CATARACT	28	0.516	30.4	203.8	ABSENT	ABSENT
5	KARIMATAN	65	M	LE	6/60	GR-II NS	32	0.505	35.2	248.47	ABSENT	ABSENT
				RE	6/12	PCIOL	32	0.533	33.2	211.33	ABSENT	ABSENT
6	KAMPAYA	69	M	LE	6/24	IMC	32	0.527	33.6	218.63	ABSENT	ABSENT
				RE	6/24	IMC	26	0.571	24.5	139.66	ABSENT	ABSENT
7	PETCHIYAMMAL	57	F	LE	6/24	IMC	22	0.565	21	121.98	ABSENT	ABSENT
				RE	6/24	IMC	22	0.564	21	122.63	ABSENT	ABSENT
8	KARUPPU	53	M	LE	6/18	IMC	26	0.622	21	108.04	ABSENT	ABSENT
				RE	6/60	IMC	32	0.636	26	124.39	ABSENT	ABSENT
9	BAKYAM	75	F	LE	6/24	IMC	26	0.622	21	108.04	ABSENT	ABSENT
				RE	6/18	PSCC	30	0.59	27.2	146.07	ABSENT	ABSENT
10	VAIYAN	53	M	LE	6/18	PCIOL	22	0.601	18.4	101.34	ABSENT	ABSENT
				RE	6/6	LC+	32	0.646	25.3	118.7	ABSENT	ABSENT
11	VELLAYAN	65	M	LE	6/12	IMC	26	0.627	20.6	105.48	ABSENT	ABSENT
				RE	6/18	IMC	28	0.516	30.4	203.8	ABSENT	ABSENT
12	NEELA	65	F	LE	6/12	LC+	32	0.533	33.2	211.33	ABSENT	ABSENT
				RE	6/6	LC+	32	0.527	33.6	218.63	ABSENT	ABSENT
13	GAYATHRI	50	F	LE	6/12	LC+	26	0.571	24.5	139.66	ABSENT	ABSENT
				RE	6/6	LC+	22	0.565	21	121.98	ABSENT	ABSENT
14	HARIHARAN	54	M	LE	6/18	PSCC	26	0.571	24.5	139.66	ABSENT	ABSENT
				RE	6/18	IMC	22	0.565	21	121.98	ABSENT	ABSENT
15	KESAVAN	52	M	LE	6/12	LC+	32	0.532	33.3	212.53	ABSENT	ABSENT
				RE	6/60	GRIINS	26	0.622	21	108.04	ABSENT	ABSENT
16	KALIDASS	65	M	LE	6/32	IMC	32	0.636	26	124.39	ABSENT	ABSENT
				RE	6/12	LC+	26	0.622	21	108.04	ABSENT	ABSENT
17	FATHIMA	54	F	LE	6/18	IMC	32	0.527	33.6	218.63	ABSENT	ABSENT
				RE	6/12	PCIOL	26	0.571	24.5	139.66	ABSENT	ABSENT
18	AMBALAM	52	F	LE	6/18	PSCC	22	0.545	22.4	135.9	ABSENT	ABSENT
				RE	6/32	IMC	26	0.571	24.5	139.66	ABSENT	ABSENT
19	RAJATHI	65	F	LE	6/24	IMC	22	0.587	19.4	108.77	ABSENT	ABSENT
				RE	6/6	LC+	26	0.561	25.2	147.26	ABSENT	ABSENT
20	BALAN	52	M	LE	6/12	LC+	22	0.435	30.1	267.27	ABSENT	ABSENT

KEYS TO MASTER CHART

M-MALE F-FEMALE

RE-RIGHT EYE

LE-LEFT EYE

BCVA- BEST CORRECTED VISUAL ACUITY

PXF - PSEUDOEXFOLIATION

U/L – UNILATERAL

B/L - BILATERAL

MLC-MINIMAL LENS CHANGES

IMC-IMMATURE CATARACT

NS- NUCLEAR SCLEROSIS

PCIOL – posterior chamber intraocular lens

IOP – INTRAOCULAR PRESSURE

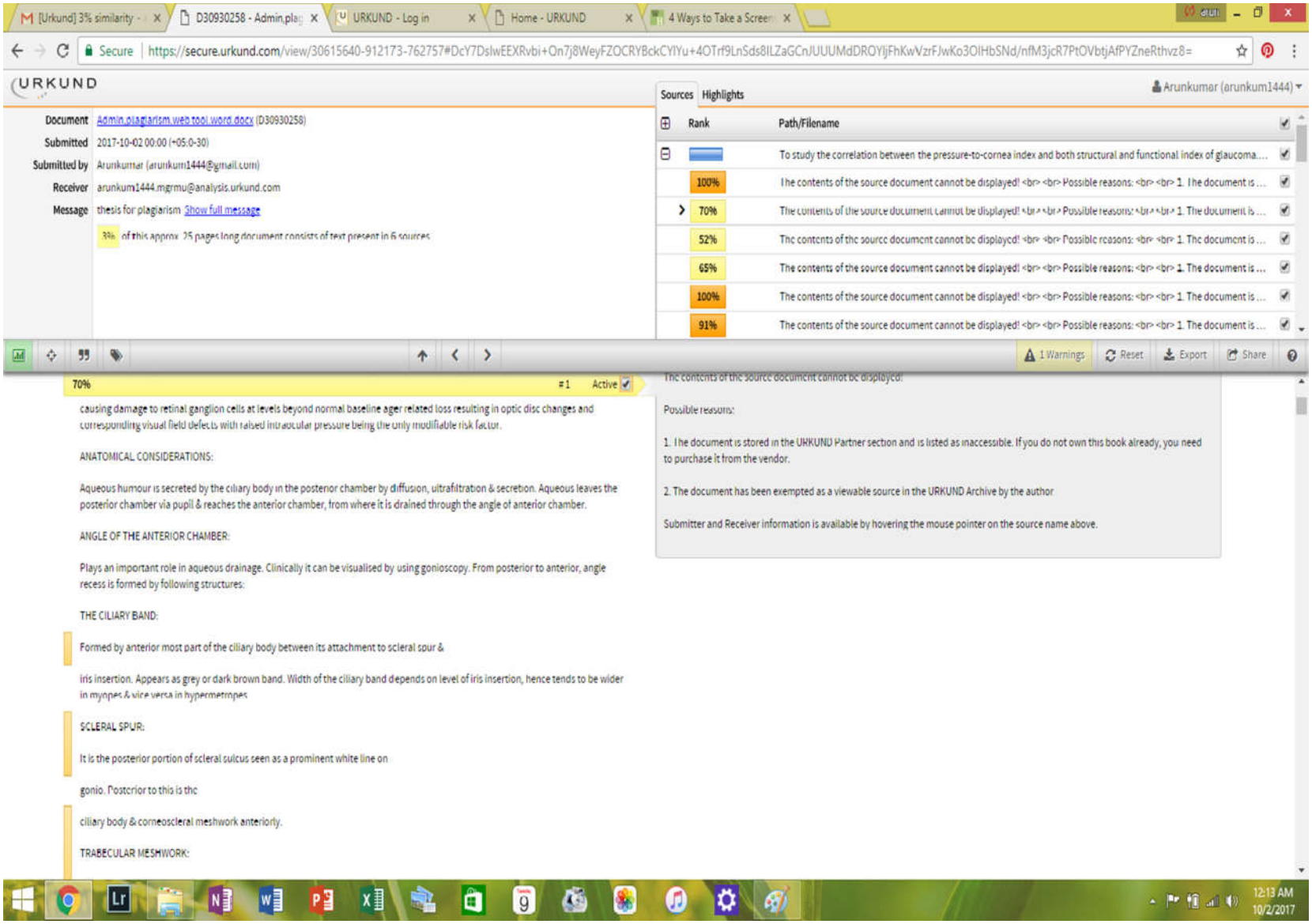
AT- APPLANATION TONOMETRY

CCT- CENTRAL CORNEAL THICKNESS

PCI – PRESSURE-TO-CORNEA INDEX

MIOP –Mean intraocular pressure.

TIOP – True Intraocular Pressure.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causing damage to retinal ganglion cells at levels beyond normal baseline ager related loss resulting in optic disc changes and corresponding visual field defects with raised intraocular pressure being the only modifiable risk factor.
ANATOMICAL CONSIDERATIONS:
Aqueous humour is secreted by the ciliary body in the posterior chamber by diffusion, ultrafiltration & secretion. Aqueous leaves the posterior chamber via pupil & reaches the anterior chamber, from where it is drained through the angle of anterior chamber.
ANGLE OF THE ANTERIOR CHAMBER:
Plays an important role in aqueous drainage. Clinically it can be visualised by using gonioscopy. From posterior to anterior, angle recess is formed by following structures:
THE CILIARY BAND:
Formed by anterior most part of the ciliary body between its attachment to scleral spur &
iris insertion. Appears as grey or dark brown band. Width of the ciliary band depends on level of iris insertion, hence tends to be wider in myopes & vice versa in hypermetropes
SCLERAL SPUR:
It is the posterior portion of scleral sulcus seen as a prominent white line on
gonio. Posterior to this is the
ciliary body & corneoscleral meshwork anteriorly.
TRABECULAR MESHWORK:
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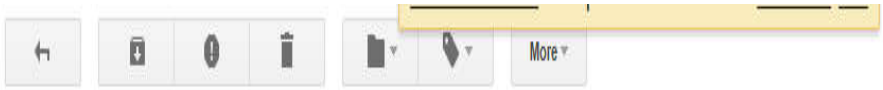

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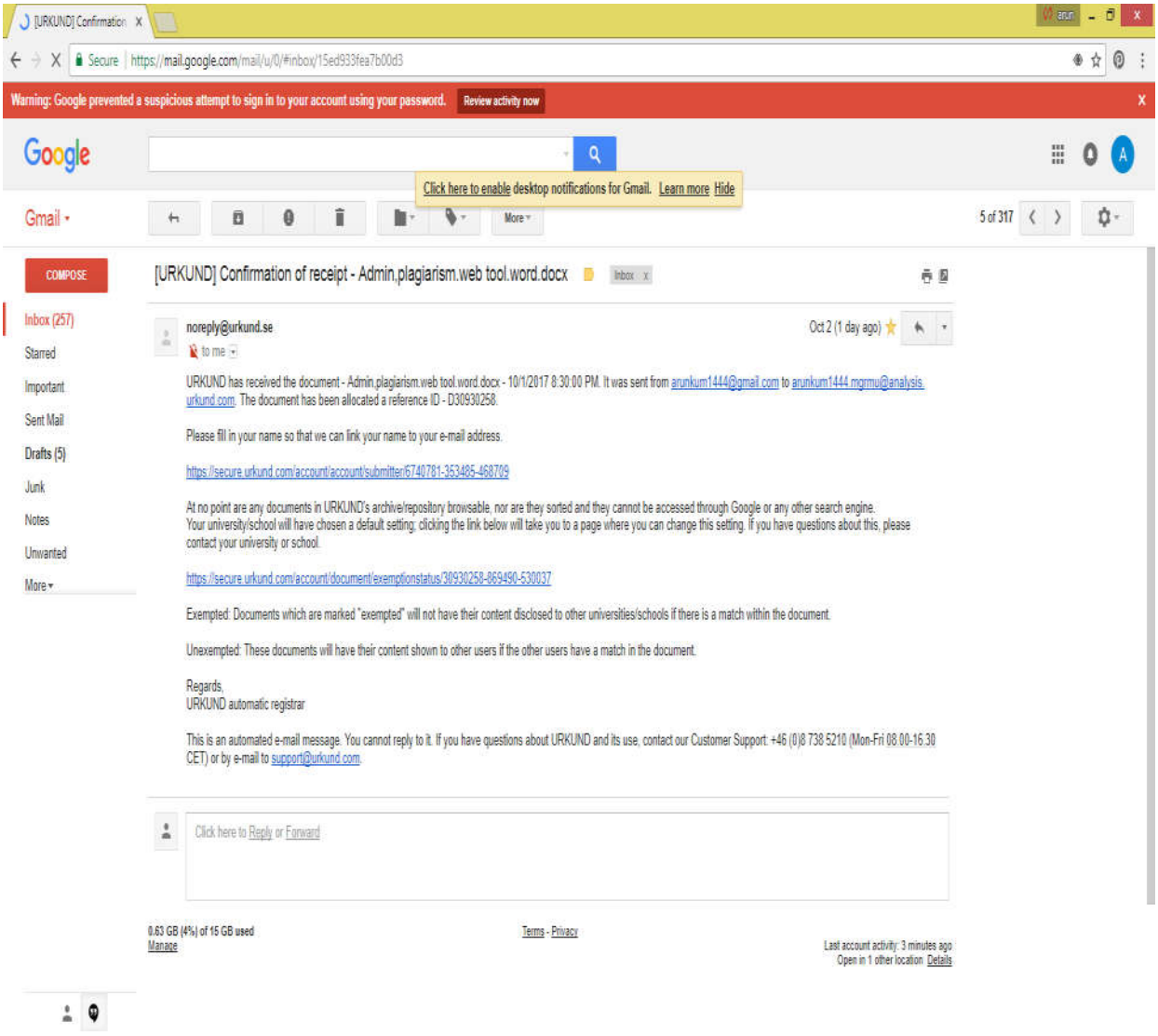
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